THE JOURNAL OF PERIODONTOLOGY

July
1938

Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY

VOLUME 9
NUMBER 2

CONTENTS

Advantages and Disadvantages of Surgical Periodontia EDWARD L. BALL . 71

Subgingival Curettage . . . . . R. W. RULE . . . 77

Evaluation of the Oral Smear in Vincent's Infection JEROME S. GROSBY . 85

The Other Side of the Story 87

Abstracts and Extracts of Current Dental Literature for 1937

(a) Periodontal Histo-pathology 88

(b) Periodontal Bacteriology 90

(c) Other Laboratory Researches 91

(d) Clinical Etiology 95

(e) Symptomatology and Diagnosis 102

(f) Treatment 106

(g) Home Care and Prophylaxis 109

(h) Vincent's Infection 111

(i) Miscellaneous 113

Twenty-Fifth Annual Meeting 119

Index of Abstracts 121

Index of Authors 122

Publication in the JOURNAL OF PERIODONTOLOGY *is not to be understood as endorsement by the Journal or the* AMERICAN ACADEMY OF PERIODONTOLOGY *of opinions expressed in any article, abstract or extract. The object of publishing the various views is to present them without prejudice to the reader for his own evaluation.*

THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers, and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and practical interest which are presented at the annual meetings of that society. The proceedings of the 1938 annual meeting will be published in the January and July, 1939, issues. This number contains the abstracts of articles on periodontia that appeared during the year of 1937.

We are grateful to all who have made available the subject matter from which the abstracts and copies have been made, including the authors of the original articles, the journals mentioned in the text of the abstract section, and those who have aided in any way in compiling the material presented in this issue.

Dental Education

The true worth of all knowledge is measured by the degree to which its essence may be conveyed to others; and therefore the function of the teacher is more exalted than that of any other man. Dental education had for its earliest exemplars, and still retains for its present sponsors, men of the loftiest and purest ideals, and a profession so endowed is indeed most fortunate.

Dental Organization

Without organization any calling becomes chaos. The genius of the men who first fostered dental organizations is better realized today even than it was in the days when they labored so loyally and lovingly in our midst. All honor to the peerless leaders of the past—they "Builded better than they knew."

Dental Science

The will to know, and know without doubt or equivocation, is the heaven-born impulse which permeates the consciousness of all truly great scientific men. Dentistry has given to the world certain geniuses endowed by nature and favored by circumstance to raise the veil over some of the obscure problems of the past, and to let the light in for the sons of men to see. They are among our greatest benefactors.

Dental Art

Science without art is truth without beauty. It is the framework without the grace and glory of color and form. "A thing of beauty is a joy forever," and what gives perpetual joy is a perpetual blessing. To bless is to perform one of the highest functions of life, and add to the happiness of the race. Without art the world would be drab, drear and dreamless.

—Dr. C. N. Johnson

Extract from the Pageant of Dental History—ONWARD.

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED THIRTY-EIGHT

Advantages and Disadvantages of Surgical Periodontia

By EDWARD L. BALL, D. D. S., F. A. C. D., Cincinnati, Ohio

WHEN a full time practitioner of periodontia is asked to prepare a discussion on a certain phase of periodontal treatment, he first considers the good results he is having in his own practice as well as those seen in the mouths of patients from other dentists, and second, the failures and discouragements he has experienced.

He then investigates the literature on the subject and evaluates the authors. In this way he can check and re-check his work, and gain confidence that the treatment is either sound and generally accepted, or *vice versa*.

At this time may I express my appreciation to the Journal of Periodontology and the Library of the American Dental Association for abstracting and collecting the literature on periodontia.

While reviewing one article after another, I became more and more discouraged because most everything I had in mind to say had already been said, so that I can only repeat and emphasize some of the salient points regarding advantages and disadvantages of surgical treatment in periodontia.

The treatment of periodontal disease may be divided into two parts, (1) Non-surgical and (2) Surgical. These two classifications may be further divided as follows:

NON-SURGICAL	SURGICAL
Medication	Subgingival curettage
Correcting careless dentistry	Flap operation
Correcting dietary errors	Gingivectomy
Relieving trauma	Electro-coagulation
Radiation	
Effective mouth hygiene	
Removal of abnormal constitutional influences	

When deciding upon the method of treatment, non-surgical, surgical, or a combination of both, it is logical and necessary to consider all available diagnostic data. After having made the necessary clinical and radiodontic examination of a case and obtaining all of the past history possible, classify the disease.

Read before the American Academy of Periodontology at Atlantic City, N. J., July 10, 1937.

DENTAL LIBRARY

The classification of periodontal lesions in Rudolf Kronfeld's book on *Histo-pathology of the Teeth and Their Surrounding Structures*, in as follows: (Extracts from pp. 292-93)*

A. GINGIVITIS
B. PARADENTAL PYORRHEA
C. ALVEOLAR ATROPHY

Alveolar atrophy is further divided into 1. Alveolar atrophy as a result of occlusal trauma, and 2. Diffuse atrophy of the alveolar bone.

The three main classifications of lesions are discussed from the following standpoints: a. Clinical manifestations b. Etiology c. Pathology d. Therapy.

A. GINGIVITIS

Dr. Kronfeld states that the therapy for gingivitis is removal of etiologic factors, scaling, and proper use of the toothbrush. We are probably all in accordance with this treatment of gingivitis, but there will always be a difference of opinion on the treatment of "paradental pyorrhea," (suppurative gingivitis, or pyorrhea, so-called).

B. PARADENTAL PYORRHEA

a. Clinical manifestations:

1. Deep pockets—sometimes one, sometimes many
2. Depth of pocket from several millimeters to entire length of root
3. Discharge of pus
4. Moderate amount of calculus usually present in pocket
5. No pain except with lateral abscess

b. Etiology:

1. Habitual lack of oral hygiene, especially in the interproximal areas
2. Mechanical deepening of pocket (toothpick, food, etc.)
3. Lateral stress in one direction
4. Systemic causes

c. Pathology:

1. Rapid apical proliferation of the epithelial attachment, and extensive detachment of the periodontal tissues from one side of the root; otherwise the periodontal membrane is healthy.
2. Wall of pocket is formed by inflamed connective tissue with epithelium covering the outer side, and ulcerated surface facing the root.
3. Inflammatory destruction of alveolar bone around the pocket.

d. Therapy:

1. Removal of etiologic factors
2. Scaling
3. Treatment of pocket: removal of outer wall (gingival flap), by packing or excision of flap

If after a most careful diagnosis it has been decided to treat a case of paradental pyorrhea by excision of the gingivae it might be well to first consider the advantages and disadvantages of this accepted method of treatment. My experience has shown me the following advantages and disadvantages:

ADVANTAGES

- *Visual Scope (a)*
- *Accessibility (b)*
- *Thoroughness (c)*
- Time saver
- More positive, recurrence less probable
- Sound surgical procedure
- Requires less skill than sub-gingival curettage
- Patient apt to take better care
- Prepares the mouth so that effective mouth care will do its part

DISADVANTAGES

- Sudden exposure of root surfaces causing temporary sensitiveness
- Abusing the method for spectacular reasons
- Exacting an excessive fee
- Post-operative discomfort
- Patients apprehensive of surgical procedure

In periodontia we are all interested, fundamentally, in removing all infected tissue, eliminating the pyorrhea pocket, establishing a healthy mouth, improving the general health of the patient and teaching this patient to keep his mouth healthy.

In the May, 1937 issue of the *Journal of the American Dental Association* is a Symposium report of a marvelous experiment and survey sponsored and supported by the California Academy of Periodontology on the surgical elimination of gingival and paradental infections at the San Quentin Prison. Some of the conclusions reached are here quoted:

"1. Paradental tissues are a frequent site of infection and a source from which pathologic bacteria are fed to other organs and tissues.
2. Study of the paradental tissues should be routine in every physical examination, especially in chronic cases.

* Printed by permission of Lea & Febiger, publishers. Also Figures 7 and 8, p. 321 from same book.

3. Failure to eliminate paradental infection is a grave over-sight."—(Oviedo.)

"1. The symptoms of about 75% of the patients were alleviated after surgical treatment of paradentosis.
2. We could find no clinical evidence of systemic damage to the patients following the surgical treatment of paradentosis.
3. There must be a very close relation between paradentosis and systemic conditions.
4. Paradentosis should always be regarded as a source of danger to the general health of the patient."—(Sweigert, Gobar, and Bartlett). (J. A. D. A. 24 (5):698, 702.)

Personally, I feel more certain of my results from gingivectomy by the abc's of the advantages namely, visual scope, accessibility and thoroughness. They assure and facilitate the complete removal of subgingival deposits, granulation tissue, sharp edges of bone, and hypertrophied gum papillae. The latter condition is especially evident in chronic or recurrent Vincent's infection.

Figure 1—Suppurative periodontoclasia with deep pockets containing granulation tissue. Heavy deposits on root surfaces. Would respond beautifully to gingivectomy.

Figure 2—Radiograms of above case showing heavy deposits and depth of pockets.

Franklin said "The eye of the master will do more good than both his hands." Cervantes in Don Quixote said "What I can see with my eyes I point out with my fingers." I firmly believe that the mistakes I have made when limiting my surgical treatment to sub-gingival curettage were made because I could not see what I was doing.

Figure 3—Marked amount of bone resorption on mesial of cuspid and first bicuspid, suppuration, no deposits. Cuspid extracted, sharp point of bone removed, thereby eliminating pocket on mesial of bicuspid. Note filling in of bone in extracted area within 3 months.

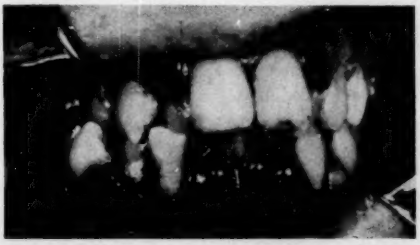

Figure 4—Chronic Vincent's infection. Note hypertrophied papillae.

The anatomy of the gums and teeth themselves make sub-gingival curettage a difficult operation especially in the concavities on the mesial and distal surfaces of most of the teeth. It is difficult to remove the hard calculus when we can see it and it is very important to avoid excessive instrumentation when there is no calculus. (Fig. 5.)

Figure 5—Showing concavities on the mesial and distal of an upper molar. Hard calculus and granulation tissue on mesial and distal surfaces.

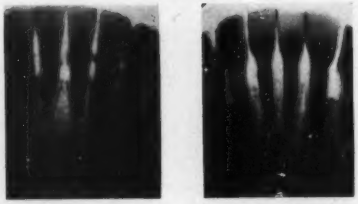

Figure 6—Suppurative type with and without calculus.

Figure 8—Higher magnification of the labial surface of the tooth. Gingival margin, ulcerated wall of the pocket; inflammation, proliferation of epithelium and alveolar bone.*

The following quotations are from authorative sources:

"Surgery is the best known method for eradicating pathologic factors in the human body; consequently, periodontoclasia cannot be an exception to this rule as its etiologic factors are located in the supporting structures of the teeth." Olin Kirkland.

"As regards method, scaling may be satisfactory except when the alveolus is involved since it is unusual to secure obliteration of the bony pockets." He contends the surgical elimination the ideal one, although he admits that drastic scaling may sometimes accomplish the results desired but this at best is an uncertain method. Karl Humphreys.

"The surgical elimination of the pocket is much simpler and requires less time." M. H. Garvin.

"Bacteria possess the power of penetrating gum tissue and with their role of foci of infection in connection with infected apices, a treatment of more than root curettage is called for." Thomas B. Hartzell.

Dr. Kronfeld was once asked this question concerning a case similar to Figures 7 and 8: "I would like to know whether by the conservative method of treatment (subgingival curettage), a healthy condition could be produced around this tooth provided that the pocket did not change markedly in depth? Would the inner wall of the pocket so heal over as to make it a safe risk for the patient?"

Figure 7 — Paradental pyorrhea on the labial side of an upper cuspid, pocket about seven mm. in depth; on the lingual surface the depth of the pocket is zero.*

Dr. Kronfeld answered, "From the microscopic aspect of this pocket, it does not seem very promising or very probable that the lower part, the distinct pouch with the narrow entrance would heal or would ever be anything else but an area of chronic inflammation. We know from examples in general surgery that whenever a pouch has been established with a narrow entrance and wider bottom, with no access for cleaning, there is always a possibility of recurrence of inflammation. I do not doubt that by dilating the pocket and packing, it would make some improvement, perhaps a complete epithelialization, and then there would certainly be no objection against the retention of the flap overlying the pocket. But on the other hand I believe there would always be the possibility of a plugging up of the drainage and perhaps a sudden flare-up of a lateral abscess, in such a case."

Dr. Kronfeld suggests lateral occlusal stress as the etiology and the removal of the outer wall to the very bottom of the pocket as the therapy.

*See bottom of p. 72.

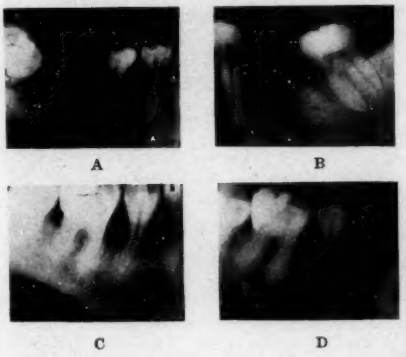

Figure 9—A few difficult cases we see frequently and which I believe are deserving of gingivectomy. A.—Lingual circular pocket. B.—Pocket on the mesial of lower molar. C.—Infiltrating type with sharp edges of bone. D.—Post-operative roentgenogram of C.

Figure 10—(Case 1.) Case of chronic Vincent's infection. After treating repeatedly for acute conditions it was decided to operate this case.

Figure 11—Ten days after operation.

Figure 12—Same case six weeks after operation. Mouth prepared for careful mouth hygiene.

Case 2—(Fig. 13)

Patient—female—age 35.

Chief complaint was an ulcerated area over the right upper cuspid and lateral and two smaller ulcerated areas on the lingual surface of the right upper jaw just opposite

Figure 13—(Case 2.) Case associated with tuberculosis.

the bicuspids. Patient gave a history of having the ulcerated areas treated for a year. Smears of the area showed spirilla and fusiform bacillus. We saw the case in September, 1935. Anti-Vincent's treatment did not give the desired result. The tissue was excised about a half inch beyond the margin for biopsy for we were suspicious of tuberculosis. Unfortunately the tubercle was not found in the specimen. The pathologist did not give us any light on the case. The blood was negative. Eight months later the tonsils were removed and sections were made. These sections showed tuberculosis. The patient died May, 1936 of miliary tuberculosis. Cases of this kind should be a warning to us that although it was almost a true picture of Vincent's, there can be other serious conditions.

Case 3.

Patient—male—age 50.

Chief complaint was a heart condition and partial vision in both eyes. A bilateral cataract operation had been performed. After all of the upper teeth were extracted the patient was quite reluctant to lose the remaining lower teeth. At that time he was despondent about his general condition. Miss Thompson of the Cincinnati Association for the Welfare of the Blind asked me if anything could be done for the remaining lower teeth. Radiograms were made (Fig. 14).

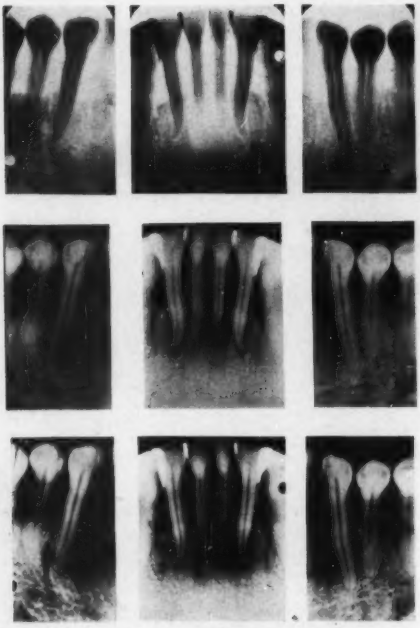

Figure 14—(Case 3.) Radiograms show a marked amount of alveolar absorption. A.—Films too light. B.—Films too dark. C.—Films about right. Good radiograms mean easier and better diagnosis.

Figure 15—Case treated by electro-coagulation. March 27, right side one week after treatment; left side four days after treatment.

Figure 16—April 12, ten days later. Note deep heavy calculus.

Figure 17—May 3, three weeks later.

After examination it was decided to electro-coagulate the infected tissue. The first photograph, (Fig. 15), was taken on March 27th. The second picture, (Fig. 16), was taken April 12. The last, (Fig. 17), May 3.

(Case 4)

Patient—male—age 45—weight 340.

Chief complaint was varicose veins and extreme pain in his legs. Had been away from his work for a month. With the exception of suppuration around the gum line, his physician could not find any other foci of infection. Radiograms showed alveolar resorption but no apical pathology. Clinical examination showed suppuration on the labial, lingual and inter-proximal areas around all of the upper and lower teeth. After pre-operative treatment the left upper and lower sides were operated surgically and the right upper and lower sides were treated by electro-coagulation. It was interesting to note that the pain in the patient's legs practically subsided after the first treatment on the upper jaw and he was able to resume work.

These photographs above shown illustrate the actual results of my work with gingivectomy.

With your indulgence let me put forth a plea for an open minded attitude with regard to this work. May I have the temerity to state that medical history from beginning to end is a glowing example of ideas rejected too hastily only to be rediscovered fifty to one hundred years later and their validity proved. Although in no manner do I wish to compare this work with the outstanding discoveries in our field. However, the mere fact that absolute truths originally propounded by, Pasteur, by Roentgen, and

others, required years before their acceptance, show us that concepts of lesser magnitude are more likely to be negated unless viewed with an open mind. The fields of bacteriology, pathology and physiology which have chronologically forced themselves to the fore in medical history are replete with instances where victory of fact has been lost for years because of the prevailing haze of prejudice. This same history of medical progress finds its counterpart in periodontia. Relatively speaking, men in recognized positions—commonly called the "tops" in their line—have been the most frequent offenders in the policy of the open mind. Because of their influence in the profession they have erroneously swayed many by their attitude and consequently have delayed the realization of new facts. A review of the history of periodontia proves this statement.

Today the advisability of surgery is being contemplated. Let us not turn our backs on the results of tried cases where these results have proved advantageous, beneficial, and successful. Let us not close our minds to new material unfolding itself before our eyes. Let us accept proved methods of treatment and hold fast to all things that are good. Finally let us recall the principles which guided the life and work of Louis Pasteur, who in a dramatic speech before the Academy of Medicine said:

Young men, doctors and scientists of the future

Do not let yourselves become discouraged by the sadness of certain hours which pass over nations.

Do not become angry at your opponents, for no scientific theory has ever been accepted without opposition.

Live in the serene peace of libraries and laboratories.

Say to yourselves first, 'What have I done for my instructions?'

And as you gradually advance, 'What am I accomplishing?'

Do this until the time comes when you have the intense happiness of thinking that you have contributed in some way to the welfare and progress of mankind.

814 Doctors' Bldg.

Subgingival Curettage*

By R. W. RULE, San Francisco, California

It may be well to state at the outset that the object of this paper is to set forth simply and concisely the conditions with which we deal in the prevention and treatment of periodontoclasia and the method of dealing with them in order to obtain the best results in such treatment.

No claims are made of offering anything new, and it is frankly acknowledged that the principles and methods discussed, have been given to the profession before in one form or another, and if there is any virtue in bringing them before you again at this time, it is in the effort to get back to fundamental principles, tried and true, which have stood the test of many years in the fire of criticism, at a time when many in the profession have turned, or are turning aside to a sort of "New Deal" type of treatment. These new fads, which are not new at all, but have been raked up out of the discard and dressed up in new clothing or given new names, are not based on true principles of healing surgery. They appeal to the operator, and in some cases, temporarily to the patient, because of their more spectacular nature, the talking point for a large fee, the fact that they do not require much skill to perform or the patient, persevering, delicate technic which must be acquired by suitable practice in order to obtain the most favorable results possible through subgingival curettage. I refer to such methods as the ruthless cutting away of the gingival tissues and alveolar bone with knife and chisel, or the sacrifice of these tissues on the altar of pseudo-science by means of the electric or chemical cautery.

And what suffering, misery and dis-

* Read before the American Academy of Periodontology, July 10, 1937, Atlantic City, N. J.

appointment to the patient! Case after case of these conditions come to our clinic as a last recourse for relief, a relief which we find difficult to afford them.

I have not seen a case in which anything approaching good results have been obtained, yet in which the pockets were so shallow that from three to five hours of careful apoxesis would have procured the finest possible results without any loss of tissue or disfigurement and practically no discomfort to the patient.

I have had patients come to me within two months of the time that the gingival tissues had been cut away and there were still pockets ranging from four to twelve mm. in depth. These were evidently among the few cases that are undertaken when deep pockets are present and no doubt the operators would have been criticized by the advocates of this method of treatment for their inexperience or lack of judgment in operating the cases. On the other hand I have seen cases operated by this method in which there were no pockets more than three or four mm. deep and in which the mutilation seemed to me nothing less than criminal malpractice.

What is to be said in defense when a supposedly scientific profession permits such methods to be taught in its schools? Forgive me this seeming tirade against such a system, but it grows out of deep feeling, not against the individual who does the work, but against the system.

One other reason for presenting the subject in answer to invitation, is the hope that in the case of some who have attempted to use the conservative methods without much success and have become discouraged, there may be something in the paper which will encourage them to make another effort and which may help them to overcome the difficulties they have encountered.

My views on this subject coincide so fully with those of T. Sydney Smith, who has been a member of this Academy for many years and who has done such splendid work in this field, that I find it exceedingly difficult to put them on paper without stating, almost in the same language, things which he has written long ago, and while I do not quote him, I have listed a number of his papers in my bibliography, and am content that he should have full credit for anything which may sound like the language of his papers. I only wish to state that I had obtained my first organic unions of soft tissues to root surfaces before I first met him or had read any of his papers, only two or three of which had been written at the time, and that we have been fast friends and advocates in a common cause since the day we first met, now about twenty-five years ago. He was the pioneer in this field and I owe much to my association with him, and believe he is entitled to much credit from us all for the splendid work he has done.

For want of time I pass the subject of etiology entirely but have mentioned prevention because such an essential part of the preventive treatment is done subgingivally, and because of its extreme importance. If the profession at large were more awake to the possibilties and value of preventive work in this field, not only would there be less curative work required but many teeth would be saved which now are being sacrificed to disease. Although diseases of the gingivae and paradentium are so extremely prevalent, prevention is not only possible in the great majority of cases but is actually simple and easy to accomplish if undertaken early enough. Volumes have been written and one might think that so simple a subject would have been exhausted. Perhaps it has; but in the main, our profession is still lethargic concerning it. There is one thing I must not pass over without mention, in regard to general operative procedure. That is the restoring of the proximal contour and each proximal area to a perfectly smooth, unbroken surface as nature intended it to be, with tight well-formed contact with the adjoining tooth. [1 2 3] This in addition to its value in preserving the teeth themselves, is a *sine qua non* for the health of the interproximal tissues.

The gingival sulcus or the inner surface of the gingival tissue at this area is the point of attack, from a local standpoint, of periodontal disease. [4 5 6] This normally shallow area between the tooth surface and overlying gingival tissues, under the

irritating effect of food accumulations and the bacterial flora of the mouth, becomes a lodging place for such debris, the soft tissues lose their healthy tone, the circulation is disturbed, the serous exudate is increased and calculary deposits begin to form. This early stage of disease is so commonly overlooked that it is the shame of the profession. From this point, progress is slow or rapid according to the conditions that pertain to the individual case.

These gingival sulci should be carefully explored in the mouth of every patient who presents for treatment. All soft debris and deposits of calculus should be removed thoroughly but gently, being very careful not to cause injury to the gingival tissues and their attachment to the tooth. This should be followed at the same or a subsequent sitting by a thorough polishing of the tooth surfaces down to the point of attachment. The polishing material should be carried by small rubber cups, thin enough to spread out over the tooth surface and under the gingival tissues and do their work without injury to these important tissues.

CURATIVE TREATMENT

We come now to the principles involved in curative treatment. The terms preventive treatment and curative treatment are relative. Clinically, no arbitrary line can be drawn where one leaves off and the other begins. Practically and for the purpose of our discussion we may say that where separation of the gingival tissues from the cementum has taken place and pockets are thus formed, the case calls for curative treatment.

The importance of the question of focal infections makes either prevention or cure imperative[7]. Finding suppurative pockets about the teeth which harbor infective bacteria and thus are, or may become, foci of infection, *we may not leave them there* and claim to have done our professional duty. The condition is not *cured* until the pyorrhetic pockets are obliterated.[8]

There are four ways in which this may be accomplished:

(1) Extract the teeth which have pockets.[9]

(2) Surgically remove the separated and diseased[8] [9] tissues to the level of the floor of the pockets.

(3) Bring about the obliteration of pockets by the reduction of hypertrophy and perhaps by an atrophic process.

(4) The fourth and ideal way in which to accomplish the [8] [9] desired result is to secure an organic union of the soft tissues to the cementum, with no loss of tissue or as little loss as possible.

Let us briefly discuss these four items. Obviously, extraction should be avoided if it is possible to accomplish the result by another method. If the conditions are such that favorable results from treatment are hopeless, or if a thorough and careful effort to save the teeth has resulted in failure, then extraction should be resorted to.

Surgical removal of the tissues, except such as is necessary in skillful curettement, is also to be avoided if possible because of the serious loss of tissue with its unsightly effect and the increased difficulty of cleaning the teeth and for other evident reasons. There may be rare occasions in which the cutting off of portions of the gingival tissues is justifiable. The writer has never found a case in which he considered that it would be possible to obtain better results by excision than by conservative treatment.

As to hypertrophy, if the pockets present are the result of an hypertrophic enlargement of the tissues, they will become shallower in depth as the hypertrophy responds to treatment. When the tissues have been reduced to normal size, which takes place very rapidly under proper treatment, if pockets are still present it is because detachment has taken place and the effort should be made to obtain the results mentioned under our fourth heading. Nevertheless, failure in this may occur, and indeed is likely to until a measure of skill in the method has been attained. Still, this does not mean that a somewhat less favorable result may not take place. In response to the removal of debris and sources of irritation, when the

surgery has not been sufficiently accurate to bring about a reunion, or where there may have been reunion only part way up from the bottom of the pocket, there will likely be an atrophic shrinking back of the gingival tissues somewhat beyond the line of their normal contour, thus reducing the pocket perhaps to the normal depth of the gingival sulcus. Or this result may be obtained with repeated efforts. The tissues resume a healthy color and tone, pus ceases to form and the circulation of the blood is restored, and all without the use of the knife. This occurs in many, many cases where the pockets are of only moderate depth and in some cases even where the work of the operator has been quite skilful. It is better, of course, than leaving pockets to result in reinfections and suppurations, but is not the ideal to strive for.

What higher ideal could we have than to retain the gingival tissues, securing a reunion with the cementum and restoring them to a healthy condition once more? If the finger, or the hand, or other part of the body were wounded and became infected, we would certainly try to bring about a surgical cleansing and healing of the wound rather than to surgically remove the finger or the part involved.

To accomplish this desirable end in the conservation of the teeth and their supporting structures we should follow the principles of conservative general surgery.[10 11 12] We must have, therefore, (1) freshly exposed living cells on all surfaces which are to take part in the healing, (2) it is equally important that none of these freshly exposed cells be impaired by the introduction of foreign agents into the wound (such as medicaments, etc.,) and, (3) these surfaces must remain in undisturbed contact for sufficient time to permit the union to take place.[13]

CONDITIONS PRESENT BEFORE TREATMENT

The conditions with which we are dealing include (1) pocket formation through separation of the gingival tissues from the root surfaces, (2) the presence in the pockets of mechanical and bacterial irritation through the formation of serumal calculus and the entrance of common mouth bacteria with the addition of food debris and sometimes overhanging filling material and crown bands, (3) also, there is an indipping of the mouth epithelium along the pocket surface as fast as it progresses, [14 15] although in some cases the epithelial tissue is broken up into islands or patches with an inflammatory condition of the connective tissues present[10 14]. I have found these cases to respond well to treatment and am of the opinion that in many of them, there is granulation tissue present in some parts of the pocket. In addition to this, we may have hypertrophy present, or there may be acute conditions with the formation of lateral abcesses and the development of sinuses as these merge again into the chronic state.

HEALING CONDITIONS

Conditions favorable to induce healing call for:

1. Removal of all irritants.
2. Cleansing of the wound.
3. Surgical freshening of the surfaces of the tissues involved.
4. Sterilization.
5. An organized blood clot.

Let us consider each of these in some detail.

1. The removal of irritants is to be done without packing away the soft tissue with cotton or other substance as has been advocated by some. Also without lacerating the tissues with clumsy instruments or careless instrumentation and without the use of chemical agents. Any of these things would militate against success. Food debris is removed with the instruments and often a considerable amount of pus may be expressed before actual scaling is begun. The calculus is then broken up and removed, and very thorough, careful scaling is done, guided by the sense of touch, upon which the mind should concentrate while at work, keeping always in mind, too, the thinness of the cementum and the important part its condition plays in the healing.

If cavities, ill-fitting crowns or faulty fillings are present, these should be attended to promptly and the teeth restored to proper form with tight, well-shaped contacts and smooth proximal surfaces, as we cannot expect to maintain the results of treatment while such conditions remain, permitting the constant packing

of food and other debris. In many cases these matters should be attended to before scaling is begun.

2. Cleansing of the wound must take place but much depends on the manner in which this is accomplished. Some have advocated the use of acids to dissolve the remaining particles of calculus and thus make up for our shortcomings in the scaling, but this obviously is opposed to the principles of healing which we are considering. Experience seems to show, as Smith has pointed out, that not even water should be used to wash these pockets after the calculus has been removed, so delicate are the requirements of nature. In many cases the tissues will be so congested that they will bleed freely at the slightest touch and this will continue until the vessels have become disengorged. This aids in the cleansing of the wound since it carries away many of the bacteria not only from the pocket itself but from the tissues also. As the work continues and the vessels become less engorged, the hemorrhage reduces and as the blood begins to clot it enmeshes the loosened particles of calculus, and these and the clot are removed together by the instrument. In cases where there is less congestion, and therefore less hemorrhage, there will nevertheless be some bleeding and it helps to serve the same purpose.[18]

3. Surgically freshening the surfaces of the tissues is essential and the cemental surface is perhaps the most important. A healing could no more be expected here without such freshening than in an old infected wound in some other part of the body. The principle is the same. Pus cells and debris have long been present and the mouth epithelium has grown down into the pocket. These conditions must be dealt with according to biological principles if we expect a biological union to take place. Surfacing of the cementum is done with freshly sharpened instruments, and light, well controlled strokes, going carefully over the whole surface involved in the pocket, right down to where the soft tissues are attached, but being careful not to sever the attachment or go any deeper. Care must be used not to cut any grooves or deep scratches in the cementum or to take away enough to expose the dentin. I cannot agree with Box [20] that "the cementum must of necessity be frequently removed in its entirety in certain locations," and that "in this event the outer dentinal layer in this region, where pockets originate and develop, presents to the curetted soft tissues a * * * suitable medium for reattachment." The experience of clinicians who have been quite successful in this work has been that where much dentin has been uncovered any organic union that has been obtained has been very slow and the result of repeated treatments and is probably the result of new cementum forming at the edge of the uncovered area.

In this important step of surgically freshening the surface of the cementum some experience is necessary to develop the judgment and to know when this part of the work has been well done. It is advisable for beginners and those who have had difficulty or failure to get results, to confine themselves at first to simple preventive work and mild cases of marginal and preparadentotic gingivitis, cultivating a patient thoroughness of technic and developing a delicate sense of touch. The tissues will be found to respond in such a way as to make both patient and operator happy. The patient's attention to and interest in the improvement which takes place in his own mouth should be enlisted. It will make him more appreciative of your work and more attentive to his own welfare in the future care of his mouth.

Little by little, more advanced conditions may be undertaken and the results will be according to the thoroughness of the work and the skill with which it is done.

As to the bone, we should not often come in contact with it since it will be resorbed in advance as the pocket increases in depth[19]. However, its cortical layer will be gone in the region of the pocket while the disease condition is active, and in some cases the instrumentation may go deep enough to encounter the bone and there may be rough or sharp edges to be smoothed up, or softened, infiltrated areas found where curettement will aid in getting results.

4. The soft tissues. These must have suitable attention. The mouth epithelium will have grown down to cover or partially cover the inner surface of the soft tissue wall of the pocket. Where the inflammatory condition is active, the connective tissue beneath will be exposed, [20] but there will at least be patches of epithelium. Just how much of this is, or should be, or can be removed in any given case is problematical. Certainly not all of it can be removed and yet results are gotten in spite of this fact. The surface at least must be freshened, that living cells may be left as a foundation for new tissue to form.

With curettes of a modified younger type, keenly sharp on both edges of the blade, while the inner edge is in contact with and surfacing the cementum the outer edge of the blade will, at least part of the time, be in contact with and lightly curetting the soft tissues. The degree to which this will be accomplished will depend somewhat on the angle at which the blade is held to the root surface. Perhaps the best angle is about 45 degrees for the major portion of the work and this angle, with a properly shaped, thin edged blade will give a sufficient curettement in most instances. Where there is much pus or inflamed tissue present there does not seem to be much curettement required.

In actual practice, many cases have responded nicely with no further attention to the soft tissues than that above mentioned. However, it seems best to go over these at the last and see that they have had sufficient preparation. Care should be used not to do any unnecessary cutting or bruising of these tissues or cause more than slight hemorrhage beyond the freeing of the congested blood vessels.

A word should be said here in regard to the use of sodium sulfide as advocated by McCall [20] and others. If this chemical agent is to be used, it should not be forgotten that it will have a detrimental effect on the tissue cells in regard to reunion, if it is used after the surgical currettement is done. Smith has suggested [27] and I believe that he is right, that if this method is to be resorted to for the removal of epithelium, it should be done before the curettement and a sufficient time allowed for the inflammation to subside. It has been stated that new epithelial tissue will form very rapidly. This is true but if the instrumentation is undertaken at the proper time, the new epithelium will be so thin and delicate that it will be easily broken up by the treatment just described.

Sterilization. Destroying or getting rid of all the bacteria would naturally be desirable, but it cannot be accomplished by artificial means. No antiseptics or germicides are to be used[21 22 23 24] etc. Nature has her own way of accomplishing this by a more certain means than we could supply. The tissues have been in a state of chronic infection and from the beginning of this, the blood and fixed cells have been building up a resistance and fortifying themselves against it with antibodies. These antibodies are a provision of nature for combating bacterial invasion, developed on provocation as an opposing force. They are substances in the blood serum or other fluids of the body, which exert a specific restrictive or destructive action on bacteria and other noxa, or neutralize their toxins.

These antibodies and phagocytic cells hold the bacteria in check and undoubtedly help to account for the fact that paradental diseases are usually slow in developing.

Now when we have given the tissues the proper surgical assistance by mechanically removing every source of irritation and greatly reducing the number of bacteria present, these natural forces quickly destroy the remaining organisms which are left in the wound and in the tissues. The rapid healing which follows the operation under favorable conditions would seem to be proof of this. Smith has stated these facts before in a number of his papers and I only review them here because of their extreme importance.

The blood clot. The importance of the blood clot has been stressed again and again.[28] It is nature's medium for forming new tissue. Without its aid we could not get these tissues to reunite. When the clot has become organized, new fibres begin to form and new tissue grows, which, if other conditions are favorable, becomes the me-

dium of reuniting the soft tissues to the cementum. When all surfaces involved have been fully prepared and the wound is clean, a little fresh blood should be permitted to enter the wound and form a clot which should then remain undisturbed. In very extensive pockets, a light pressure of the finger over the area of separation for the moment required for clotting may aid in the clot forming and holding the tissues in apposition to the root surface[20].

If these principles of treatment are followed carefully, results will be obtained which will be in proportion to the skill and thoroughness of the technic employed. If everything has been done just right, a reunion will take place within from five to seven days and with reasonable care thereafter, it will remain permanently firm and sound. There has been much discussion of this point. Why should it be thought incredible that such results should be obtained routinely by those who have given much time and practice to this work? Many histologists agree that it is biologically possible. Some of them have produced what they consider histologic evidence. Many clinicians have produced and continue to produce what they and their associates consider quite definite clinical evidence. The tissues in these cases have remained healthy and sound without re-infection over periods of many years and continue to so remain. It is difficult to obtain specimens for histologic work. Skillen and Lundquist[20] recently presented a report of some studies in this field in which they conclude that reattachment in human material is improbable. They present evidence of some reattachment in animal experiment and state that the results of their experiments with human tissue were rather conclusively against reattachment, and yet their methods of treatment were such that we clinicians would not consider them an adequate test.

Several cases are on record, of which one reported by Kronfeld[21] is the most recent, showing how new cementum and connective tissue fibres, apparently of the true pericemental type of attachment will form to unite the broken ends of a fractured root. Here the cementum is shown to form right across the fractured ends of the dentin and somewhat into and along the walls of the pulp canal. Repair seems to have begun with resorption at the fractured dentinal surfaces followed by deposition of cementum at these areas and development of fiber bundles. Epithelial attachment to the dentin is shown to have developed on both fragments where the fracture line through the dentin ran through the epithelial attachment. True, the injury took place in these cases in such a way that infection did not follow and nature could accomplish the repair without the assistance of surgical intervention, but they show the possibility of new cementum and connective tissue fiber as well as epithelial attachment taking place under favorable conditions. Moreover, the principles of surgical healing in other parts of the body where infection has taken place, have long ago been established, and would it not seem, as some of us have contended, that failure to get results is due to our inadequately following these principles.

When pockets from ten to fifteen mm. deep cease to emit pus and the tissues become healthy and refuse to permit the passing of an instrument between them and the root without pain and hemorrhage and the decalcified, resorbing condition of the alveolar bone which has taken place under the processes of inflammation, appear roentgenographically to have given way to recalcification or the forming of a new cortical layer with the subsidence of inflammation, and the patient's health greatly improves and these conditions remain for many years without a sign of recurrence or reinfection, what has happened?

Having treated a case and obtained good results, the patient should be informed of the need for future care and should be called in at intervals of 2, 4 or 6 months for a check up and mouth hygiene treatment. On these occasions a careful examination of all the gingival sulci should be made and especially those areas which had been affected with deep pockets. If traces of pockets are found to be still present, another attempt should be made to get these to close up. The writer has been successful sometimes after the third

or fourth attempt. He is of the opinion that the more one does of this work, the better results one will get and the greater will become his interest in the work. Certainly there is no greater service that the dental profession can render than first, to prevent these diseases in as great a number of patients as possible, and second, to cure them for those who are unfortunate enough to be afflicted with them.

BIBLIOGRAPHY

1. Wedelstaedt, E. K.: Unpublished notes.
2. Black, G. V.: Beginnings of Pyorrhea Alveolaris: Treatment for prevention, Items of Interest, June, 1911. p. 247.
3. Smith, T. Sydney: Inflammatory Diseases of the Gingival Margin and Periodontal Membrane (Pyorrhea Alveolaris) Dental Cosmos, Nov., 1916, p. 1246.
4. Smith, T. Sydney: Periodontal Septic Foci, Calif. State Jour. of Medicine (Jan.), 1916, p. 357.
5. Merritt, Arthur H.: Periodontal Diseases, p. 21, The MacMillan Co., 1930.
6. Stillman, Paul R. and McCall, John Oppie: *A Textbook of Clinical Periodontia*, p. 65 and p. 82, 2nd Ed. The MacMillan Co.
7. Smith, T. Sydney: The Successful Scientific Treatment of Periodontal Diseases, Items of Interest, 1916, p. 438.
8. Smith, T. Sydney: The Emetin Hydrochloride Question in the Treatment of Periodontal Diseases, J. A. M. A., pp. 64: 1567, 1568, 1569, May 8, 1915.
9. Smith, T. Sydney: Constructive Treatment of Diseased Paradental Tissues, J. A. D. A. 22: 1477-1491, Sept., 1935.
10. Smith, T. Sydney: The Emetin Hydrochloride Question in the Treatment of Periodontal Diseases, J. A. M. A., May 8, 1915. 64: 1568, May 8, 1915.
11. Smith, T. Sydney: Periodontal Septic Foci, Calif. State Jour. of Medicine, Jan., 1916, p. 360.
12. Smith, T. Sydney: Constructive Treatment of Diseased Paradental Tissues, J. A. D. A. 22: 1468, Sept., 1935.
13. Smith, T. Sydney: The Successful Scientific Treatment of Periodontal Diseases, Items of Interest, 1916, p. 444.
14. Gottlieb, Bernard: Tissue Changes in Pyorrhea, J. A. D. A. 14: 2178-2207, Dec., 1927.
15. Becks, Hermann: Normal and Pathologic Pocket Formation, J. A. D. A. 16: 2182, Dec., 1929.
16. Kronfeld, Rudolf: The Condition of the Alveolar Bone Underlying Periodontal Pockets, Jour. of Periodontology, 6:25, Jan., 1935.
17. Smith, T. Sydney: The Successful Scientific Treatment of Periodontal Diseases, Items of Interest, 1916, p. 446.
18. Smith, T. Sydney: Idem.
19. Kronfeld, Rudolf: The Condition of the Alveolar Bone Underlying Periodontal Pockets, Jour. of Periodontology, Jan. 1935, p. 27.
20. Gottlieb, Bernard: Tissue Changes in Pyorrhea, J. A. D. A., Dec., 1927.
21. Smith, T. Sydney: Inflammatory Diseases of the Gingival Margin and Periodontal Membrane (Pyorrhea Alveolaris) Dental Cosmos, Nov., 1916, p. 1248.
22. Smith, T. Sydney: The Emetin Hydrochloride Question in the Treatment of Periodontal Diseases, J. A. M. A. 64: 1568, May 8, 1915.
23. Smith, T. Sydney: The Successful Scientific Treatment of Periodontal Diseases, Items of Interest, 1916, p. 446.
24. Smith, T. Sydney: Constructive Treatment of Diseased Paradental Tissues, J. A. D. A. 22: 1487, Sept., 1936.
25. Smith, T. Sydney: The Emetin Hydrochloride Question in the Treatment of Periodontal Diseases, J. A. M. A. 64: 1569, May 8, 1915.
26. McCall, J. O.: Improved Method of Inducing Reattachment of Gingival Tissues in Periodontoclasia, Dental Items of Interest, 68: 342-362, Nov. 1925.
    Stillman, Paul R. and McCall, J. O.: *A Textbook of Clinical Periodontia*, p. 220, Second Edition. The MacMillan Co.
27. Smith, T. Sydney: Constructive Treatment of Diseased Paradental Tissues, J. A. D. A. 22: p. 1488.
28. Box, H. K.: Treatment of the Periodontal Pocket, Research Bulletin, No. 10, The University of Toronto Press, 1898, p. 42.
29. Idem: p. 99.
30. Skillen, W. G. and Lundquist, G. R.: An Experimental Study of Peridental Membrane Reattachment in Healthy and Pathologic Tissues, J. A. D. A. 24: 275-285, Feb., 1937.
31. Kronfeld, Rudolf: A Case of Tooth Fracture, with Special Emphasis on Tissue Repair and Adaptation Following Traumatic Injury. Jour. Den. Research, 15: 429-446, Dec., 1936.

Evaluation of the Oral Smear in Vincent's Infection*

By JEROME S. GROSBY, D. D. S.[1]
St. Louis, Missouri

SINCE Plaut and Vincent discovered the causal relationship between ulcero-membranous gingivitis and the treponema Vincenti (Vincent's spirochete) and fusiform bacillus, much has been written. So much in fact, that the present time finds many diversified opinions regarding the diagnostic value of the smear. Opinions range from that of giving a "positive" smear no clinical value at all, to using it as the sole diagnostic sign of a Vincent infection. That the organisms are found in many normal mouths is not denied. At other times they are almost totally absent, although clinical signs may be indicative of the presence of trench mouth. They are frequently (almost invariably) present in periodontoclasia, yet there may be no evidence of an ulcero-membranous infection.

Realizing the chaotic nature of the subject, the writer endeavored to determine whether or not the picture could be clarified. The following conclusions are the result of investigation of approximately one thousand cases of normal and pathological mouths. This extended over a period of several years. Various methods of obtaining specimens and staining were used. Among many of the stains applied were, Gram's, methylene blue, fuchsin, victorian blue and gentian violet.

The technic finally developed as simplest and most satisfactory was to secure the specimen, using a nichrome wire loop, from the depth of a pocket or gingival crevice or ulcer surface (haemorrhage must be avoided). The area was first swabbed with alcohol and extraneous adhering debris removed. With a sterile loop the specimen was secured and smeared upon a clean glass slide. It was fixed by passage over a bunsen flame three or four times. After cooling, the smear (it should not be too heavy—experience is the best indicator) was covered with the gentian violet and allowed to remain for one-half minute. The excess stain is then washed off with tap water, the slide dried and examined under the microscope. The stain used was a ten per cent aqueous solution made with a saturated alcoholic solution of gentian violet (one cc. saturated alcoholic solution gentian violet added to nine cc. distilled water). The solution is fairly stable and may be kept from six to eight weeks.

Heretofore the presence of the fusiform bacillus and treponema Vincenti has been considered diagnostic. Other elements in the smear and variations in morphology of the organisms were either overlooked, ignored, or considered valueless. Herein lies the error that has been causing so much confusion. Let us first consider the morphological changes that have a bearing upon the diagnostic value of the microscopic picture. The spirochete shows little recognizable variations, but the fusiform bacillus manifests changes in form and structure which have a definite relationship to pathogenicity. In the acute stage (ulcero-membranous gingivitis) the fusiform bacillus and the spirochete coexist in large numbers. The fusiform bacillus, as its name implies, is fusiform in shape, having pointed ends and showing a definite granular structure which appears as alternate light and dark bands or beadings. There may be from one to six of these light bands in the individual bacilli. When we find these characteristics we may be reasonably sure that the organism is in a virulent state and either definitely active or potentially so. As the clinical picture becomes chronic or a mixed infection develops with a lessening of the acute signs, the picture changes. The proportion between spirochetes and fusiform bacilli is altered—the fusiform bacilli becoming relatively less in number and showing definite change in shape and structure. The pointed ends become blunt or rounded and the spindle shape is lost. The beaded appearance lessens and is often totally absent, the bacillus now taking a solid stain. The organism becomes smaller and may be found to be slightly curved or comma-shaped. Apparently this is a retrograde involuted form or perhaps a resting stage. Certainly it is one of lesser biological activity on the part of the bacillus.

* Original communication.

[1] Department of Periodontia, St. Louis University, School of Dentistry.

Another very important consideration that has been overlooked is the presence of the neutrophilic leukocyte or pus cell. In the acute stage leukocytes are practically absent. At most one or two may be found in the average field under oil immersion. As the acute stage subsides a localized leukocytosis develops and we find large numbers of pus cells appearing (these so-called pus cells or leukocytes are characteristic in most exudative inflammatory processes). This coincides with the aforementioned morphological change in the fusiform bacillus. Large numbers of granulocytes plus spindle-shaped granular bacilli are rare, but would be interpreted as a mixed infection and we would probably find large numbers of coccoid organisms. This brings up another phase of the picture. The presence of staphylococci in large numbers is commonly found with many pus cells. When, however, we have many spirochetes and active fusiform bacilli the cocci show a proportionate decrease in number.

Regardless of morphology, if large numbers of spirochetes and fusiform bacilli are found in an apparently normal mouth, we may assume that they are potentially active and only await an opportunity of lowered tissue resistance. In a well-cared for, clean mouth with normal mucous membrane they are rarely seen beyond two or three to the oil immersion field.

An interesting point of speculation that would bear research is the fact that agranulocytosis, in which the granular leukocytes are deficient in number in the blood stream, is accompanied by mouth and throat ulcerations in which large numbers of fusiform bacilli and spirochetes are found. This presents a picture similar to that seen in examination of the smear in acute Vincent's stomatitis—notable absence of granulocytes.

One other error frequently made is to diagnose a case as positive on the basis of finding large numbers of spirochetes only. It should be borne in mind that there are several types of treponema that normally inhabit the mouth, only one of which is the treponema Vincenti. They are similar in shape and size but probably bear little if any relation to trench mouth. Apparently it is the fusiform bacillus that plays the greater etiologic role and perhaps this accounts for the frequent failure of spirochaeticidal therapy.

To summarize, we may say that a positive diagnosis should depend upon finding of the treponema Vincenti and the spindle-shaped granular staining fusiform bacillus in large numbers concommitant with a relative absence of pus cells and very few coccoid organisms. With chronicity or subsiding of the infection the fusiform organisms show retrograde morphological changes and pus cells make their appearance. Expressing it as a formula we would present it as V=F/Lf where V is the Vincent infection, F the spindle-shaped granular staining fusiform bacillus, L the leukocytes, and f the retrograde form of fusiform bacillus. As a matter of record, we have never seen large numbers of fusiform bacilli without the coexistence of the spirocheta.

Quite obviously the above conclusions in order to be of any value must be predicated upon careful technic and correct interpretation of the elements in the microscopic picture. It has been found, however, that by using this refined method of studying the smear we do find a very close correlation between the clinical and laboratory diagnosis. Regardless of the value of this technic no attempt is made to make the smear the sole diagnostic factor. That would be a grave error since all factors at our disposal should always be used in making a complete oral examination and arriving at a correct diagnosis. It is hoped, however, that the above will in some measure clarify the existing doubts as to the value of the oral smear in Vincent's infection.

704-5 Missouri Theatre Bldg.

NOTICE TO MEMBERS

The Editor continues to receive requests from medical and dental libraries for the January, 1930 issue of the Journal of Periodontology to complete their files of this publication. The coöperation of members in supplying this need will be greatly appreciated. Address communications to

JOURNAL OF PERIODONTOLOGY, *555 W. Maple Ave., Birmingham, Michigan*

The Other Side of the Story

"Courage consists in equality to the problem before us." . . . . EMERSON

DENTISTS have been more fully aware of the universal need of dental health service than any other group since it is brought daily to their attention by the mouth conditions of their patients. They are ever conscious of unclean teeth, inflamed gingival tissues, evidences of malocclusion, pulpless teeth, dental caries, missing teeth, and other dental defects in the mouths of those they contact outside of office hours and of the man on the street. It is no news to dentists that only about twenty-five per cent of the population have dental services outside of extractions. That individual dentists have contributed not only time and money but their personal services to a large number of this twenty-five per cent may be news to those who have so freely criticized dentistry for its lack of social consciousness. That the advancement of dentistry has been due to the personal sacrifice in time, funds, and research by individual dentists, most of whom have been members of *organized dentistry*, may be news to them also. Throughout the country many ethical dentists have been prodigal with their time and talents in welfare and dental educational work in clinics and schools, and without remuneration. It would be impossible to record the vast amount of work done for the indigent and the semi-indigent patients in dental offices, but, it might have a quieting influence on our critics if a record were kept of the dental services contributed outside of the individual dental office by those who have been responsible for the progress of dentistry.

In spite of pretenses to the contrary, it is now quite generally, but unwillingly, believed in dental circles that any advice from organized dentistry will be ignored in the federal plan to extend the benefits of dental service. In spite of this there are certain fundamental principles which if ignored will wreck any plan or at least will sink the funds used for the purpose in a bottomless pit.

Dentists know that there is more to the problem of extending dental health service than pushing a button in Washington or in providing funds; that the removal of infection from the mouth and the relief of pain are the first and most important steps to be taken; that prevention of dental and periodontal disease is the best practical means for approaching a solution to the problem; that unless the patients receiving these services value their teeth to the extent that they will keep them clean, many fillings will fail and dentistry will be blamed; that these patients will be unable to provide their own tooth brushes and many would not use them if they could; that *quality* of service is *important*, and if ignored will discount much of the benefit received; that the number of those entering the profession each year is less than those dropping out from death or other causes.

It is important that every dentist keep himself well informed upon all legislation, federal, state, or local, relating to dentistry and social welfare, and that he coöperate with *dignity and courage* in any plan to extend the benefits of his profession. It is certain that no other minority group will be as pleased as dentistry to see its services available to all people.

—*G. R. S.*

ABSTRACTS AND EXTRACTS OF CURRENT DENTAL LITERATURE FOR 1937

Periodontal Histo-pathology, Periodontal Bacteriology, Other Laboratory Researches, Clinical Etiology, Symptomatology and Diagnosis, Treatment, Home Care and Prophylaxis, Vincent's Infection, and Miscellaneous Subjects

(a) Periodontal Histo-pathology

(Continued from 8(2):106. July, 1937)

186—Brinch, Ove: (Kopenhagen): **Ueber einige bisher unbekannte pathologische Gewebsveränderungen in der Pulpa und in Paradentium in einem Falle von Skorbut.** Concerning some heretofore unknown pathologic tissue changes in the pulp and in the paradentium in a case of scurvy, in: D. Z.-M.-U. Kieferhkde. 1937.

Dr. Brinch describes an interesting case of a 49 year old man who died of scurvy. On dissection, besides a number of other degenerations there were also found changes of the dental tissues not previously described. These occurred in the pulp of the incisors, in granulation tissue of the gingivae, in the paradental medullary spaces; they were weakest in the incisal region, more marked in the molar teeth. In the author's opinion, some material is excreted through the capillary walls which causes such changes in the surrounding tissues.

The microscopic picture showed a *circular segment which without break runs transversely through different tissues.* In the bone there was an abnormally abundant deposit of collagenous tissue in the ground substance, which is also the seat of a typical osteoporosis. New osteoblastic formation of bone is as rare as degeneration and then occurs only at the marginal borders of the alveolar process. The security of the teeth was endangered by the fact that in many places the alveolar walls were defective.

Brinch seeks to explain the origin of the coarse fibred hetero- and para-plastic bone tissue by the year round diet of the patient: bread, butter, eggs, smoked bacon, and coffee; no fresh fruit, vegetables, potatoes.

The pathologic tissue changes were those of scurvy, the traumatic effect on the paradentium, and a weakening of the vascular system.

—*Abstr. Transl. from Paradentium*
—*H. A. R.*

187—Hall, J. Frank and Hamilton B. G. Robinson: **Alveolar atrophy in anemic dogs** (Abstr. 105), in: J. D. Res. **16**(4): 345-6. Aug., 1937.

All mouth conditions charted in ten bull dogs and thirteen coach dogs from colony of anemic dogs. Two dead coach dogs and one dead bull dog also available for histological sections. Dogs lived in animal house since birth, 1-13 years, on carefully prepared and balanced soft diet, although kept anemic by repeated routine extractions of blood. Owing to continual chewing on metal cage and feed pan, anterior teeth in many cases broken and abraded, latter mobile when not in occlusion. Much calculus on practically all posterior teeth; very little abrasion regardless of age. In early stages of alveolar atrophy: hypertrophy and inflammation of soft tissue, with beginning atrophy of osseus structure, followed by extensive atrophy of both alveolus and supporting soft tissue. Microscopic examination: alveolar atrophy and many multinucleated osteoclasts, not proportionately greater in areas of greater resorption. Noted compensatory hypercementosis, followed by root resorption. Animals having been fed on soft food since birth, alveolar atrophy probably due to non-use.

—*Copy.*

188—Thoma, Kurt H., D.M.D. and Henry M. Goldman, D.M.D.: **Classification and histopathology of parodontal disease,** in: J. A. D. A. **24**(12): 1915. Dec., 1937.

The author's own summary states that the object of his report was to present new evidences of the histopathologic process in

parodontal diseases which were based on a study of sixty-seven jaws obtained at necropsy. Parodontal disease was classified as (1) simple gingivitis, (2) marginal parodontitis, (3) parodontosis, (4) the syndrome of marginal parodontitis and parodontosis. The pathologic processes of each type are described.

In simple gingivitis there is inflammation of the gingiva, with redness and a tendency to hemorrhage; no pockets and no pus present; epithelium attached to the tooth at the cemento-enamel junction, and showing slight papillary down-growth into the gingival corium. Round-cell infiltration in the connective tissue is a feature.

In marginal parodontitis, the local type of parodontal disease, the condition begins at the alveolar crest and extends downward, the first changes occurring in the gingivae and later in the bone. The epithelium proliferates and extends into the underlying gingival corium, and grows down along the cementum also. The round-cell infiltration is most marked. The peridental membrane disintegrates as the bone is resorbed. The crest of the alveolar bone usually has a pitted appearance.

Parodontosis is of systemic origin, the alveolar bone being first affected, and is a disease of that structure. The main pathologic change is bone reversion in which the bone disappears without the intervention of osteoclasts or other cellular elements. There are changes in the alveolar bone marrow and in the cementum, those in the peridental membrane being limited to the blood vessels.

The syndrome of parodontosis and marginal parodontitis which commonly occurs makes diagnosis difficult. This is fully described.

The author plans to correlate the pathology with the clinical symptoms and the medical history of the patients whose jaws were studied for this report.

—*G. R. S.*

189—Wolbach, S. B., M.D.: **Pathologic changes resulting from vitamin deficiency.** Bills, Charles E., Ph. D. **New forms and sources of vitamin D.** Youmans, John B., M. D.: **Present status of vitamin deficiencies in practice,** in: J. A. M. A. **108**(10): 7. Jan. 2, 1937.

An interesting series of papers the first in particular having certain dental and oral implications. Wolbach's histo-pathological studies of effects of vitamin A and vitamin C deficiency are exhaustive. Vitamin A deficiency shows first in epithelial structures with atrophy of the tissue and the substitution for it of a keratinizing epithelium. In the human tooth germ the enamel organ is altered so that probably this lack during the formative period of teeth outranks all other vitamin deficiencies in importance. The important gross features of scurvy at all ages are hemorrhages and changes in the bones, all of which are explainable on the basis that vitamin C deficiency causes cessation of formation and maintenance of intercellular substances. Calcium metabolism is not primarily affected. The sites of the characteristic hemorrhages in bone, muscle, skin and alveolar process are determined by mechanical factors, stresses and trauma. In gingival hemorrhages, resorbtion of alveolar process and loosening of teeth, the two factors of stresses and loss of intercellular substance are apparent. Vitamin C deficiency probably also affects forming teeth by interfering with function of the odontoblasts. Repair of lesions of both A and C deficiency occurs with dramatic rapidity on correction of the diet or, in the case of vitamin C, the addition of cevitamic acid. The role of vitamin D in metabolic processes is less clear but besides the well known effect on calcium and phosphorus utilization it is known to suspend the cartilage cell cycle essential to endochondral growth of bone, while not impairing multiplication of cartilage cells. Vitamin B is apparently directly concerned in nervous system physiology, since nerve structures return promptly to normal function with its administration, if given in time. Lesions of the nervous system seem also to be among late secondary consequences of vitamin A deficiency. According to C. E. Bills eight forms of vitamin D have been artificially prepared. At least two forms occur in fish oils which may or may not be identical with those made *in vitro*. Youmans thinks that while diagnosis may be difficult in the mild types of avitaminosis there are scientific methods available for making these determinations, and early and accurate diagnosis is important.

(b) Periodontal Bacteriology

(Continued from 8(2):109, July, 1937)

74—Appleton, J. L. T., B.S., D.D.S., and Anne K. Dietz: **In vitro effect of saliva on bacteria,** (Abstr. 59), in: J. D. Res. 16(4): 325. Aug., 1937.

Inocula of various densities of *Serratia marcescens, Escherichia coli, Eberthella typhosa, Ps. aeruginosa,* seeded into human saliva; incubated at 37° C.; samples of mixtures taken at various times (1.5, 3, 6, 9, to 24 hours). Relative number of bacteria estimated in samples. Saliva from different individuals centrifuged; some sterilized by filtration; some heated at 56°C., 30 minutes; in some bacteria, removed by centrifugation, replaced after filtration or heating. All centrifuged saliva anti-bacterial. Filtered or heated, centrifuged saliva less anti-bacterial. Anti-bacterial effect apparently restored by returning bacteria separated by centrifuging.

—*Copy.*

75—Appleton, J. L. T., L.M. Gette, and C. Douglas: **Effect of oral mucosa on applied bacteria,** (Abstr. 60), in: J. D. Res. 16(4): 325. Aug., 1937.

Suspensions of *Escherichia coli* painted on areas of oral mucosa (lingual dorsum, hard palate, cheek, mucous surface of upper lip, external alveolar plate of maxillary anterior teeth). Swabbings, taken immediately and after 3 min. (from symmetrical area), transferred to surface of plates containing sterile gentian-violet agar. After 24 hrs. at 37°C. number of colonies from immediate and 3-minute swabbing compared. Results confirmed work of Arnold *et al.:* oral mucosa somewhat bacteria-fixing or anti-bacterial, analogous to action of skin. Different areas of oral mucosa varied in frequency of effects. Smaller series, substituting *Serratia marcescens* for *E. coli:* similar results.

—*Copy.*

76—Beust, Theo. B., D.D.S., M.D.: **Micrology of gingival inflammations,** in: J. D. Res. 16(2):157-61. Apr., 1937.

Author's summary:

"1. The *Spirillum sputigenum* should be ranked as an important factor in oral inflammations.

2. The nomenclature applied during W. D. Miller's time and shortly thereafter should be retained until exhaustive morphological studies justify a change. Changes in terminology, in the present state of our knowledge can be only temporary.

3. *The therapeutic use of autogenous vaccines made from cocci grown from lesions in which non-cultivatable mouth organisms abound, appears illogical and unscientific."*

(Italics not in original.)

—*Copy.*

77—Bibby, Basil G. and Rita P. Ball: **Effect of saliva on growth of bacteria,** (Abstr. 61), in: J. D. Res. 16 (4): 325. Aug., 1937.

Graded inocula mixed in agar-pour cultures; plated; saliva added to wells cut in hardened agar. Growth-inhibitory effects manifested as colony-free zones about wells. Study included 17 types of bacteria; 117 strains. Each type subjected to action of at least four different salivas. General bacteriostatic effects: marked action against organisms not usually present in mouth; slight or absent against organisms normally present.

—*Copy.*

78—Grythe, Olaf: **Die Bakteriologie der Paradentose.** The bacteriology of periodontoclasia, in: Norske Tidende. Aug., 1937, and Zahnärzt. Rundsch. 49: 2164. 1937.

No single group of bacteria plays a unique role as etiologic factor in periodontoclasia, but certain bacteria contribute to its direct causation through their toxins and thus endanger the periodontium. As indirect causes they create favoring conditions by tartar and plaque formations.

Since our knowledge of the great variability of the bacterial flora of the oral cavity is limited, Grythe suggests making a basis for hypotheses by investigating the bacterial flora of the child's mouth; a comparative study of the oral cavity of adult individuals will then clear up the different microbic varieties and render possible a better evaluation of the significance of the bacteria, in the pathogenesis of oral cavity diseases.

—*H. A. R.*

79—Hood, M. and Arnold L.: **Relation between the oral and the gastric flora.** American Journal of Diseases of Digestion and Nutrition. Mar., 1934, p. 40. Abstr. (abbreviated), in: J.A.M.A. 108(19): 1675. Mar. 8, 1937.

Similarity between stomach and oral bacterial flora would indicate that the oral flora

is either a transient or a regular inhabitant of the stomach and while these organisms are mostly non-pathogenic, virulent kinds are sometimes present thus adding to the load of infective material which must be taken care of by the disinfecting power of the gastric juice. In attempting to reduce the quantity of bacteria passing into the stomach from the oral cavity it was found that physiologic salt solution was as effective as any rinse in reducing bacterial count, the mechanical washing apparently being the effective agent rather than the bactericidal value of the solution. The quantity of bacteria in the stomach, however, was not noticeably altered by this oral washing nor was it appreciably affected by diverting the saliva from the stomach for as long as two hours.

—*Copy.*

80—Rosenthal, S. Leonard, D.D.S.: **Effects of medicaments on the motility of the oral flora,** in: J. Periodont. 8(2):71-80. July, 1937.

Author's summary:

A new test of antibacterial efficiency is described.

Sixty-eight solutions recommended for the treatment of Vincent's infection are evaluated.

The action of chromic acid is purely caustic. No oxygen is evolved when chromic acid is applied to the tissues.

Nascent oxygen is of no value in the treatment of Vincent's infection other than mechanical cleansing.

Arsphenamine is not only a spirocheticide, but is efficient against the entire oral flora.

Among those substances which are not injurious to the oral tissues, those instantly stopping movements of the entire flora are: 10% aqueous solution of arsphenamine, 10% arsphenamine diglucoside, 25% aqueous solution of iodine, Talbot's iodoglycerol, tincture of metaphen, and 1.5% aqueous solution of bismuth sodium tartrate. Slightly less efficacious but still of value are: saturated solutions of copper sulphate and Mapharsen, 20% argyrol, and Lugol's solution.

Most efficient against the non-motile flora and not injurious to the tissues are: Lugol's solution, 25% aqueous solution of iodine, Talbot's iodoglycerol, and tincture of metaphen (untinted). Markedly affecting growth, but with a few colonies surviving are 10% aqueous solution of arsphenamine, 10% arsphenamine diglucoside, saturated solution of Mapharsen, and tincture of metaphen (tinted).

—*S. L. R.*

81—Weiss, M. ——: **Contribution a l'étude de la paradentose,** in: Revue de la Société Cubana. Oct., 1937, pp. 56-68.

In a well-documented article, the author stresses the usefulness of microbic cultures in each case,—not only for the microscopic examination of the microbic flora of pyorrhea, but also for the sake of observing the perceptible bacterial diminution as the treatment progresses. This procedure permits the following up of the action of various medicaments and the determination of those bacteria actually most resistant to treatment.

The media used are simple agar, blood agar, or blood and bouillon—agar.

—*H. A. R.*

(c) Other Laboratory Researches

(Continued from 8(2): 110, July, 1937)

70—Boyle, Paul E., D. M. D.: **Supporting structures of incisor teeth of guinea pig in experimental scurvy,** (Abstr. 44), in: J. D. Res. 16(4):317. Aug., 1937.

Scurvy so weakens peridental structures of guinea pig that incisor teeth become loose and easily extractable. X-ray and histological examinations reveal marked rarefaction of alveolar bone and widening of peridental membrane. Changes more conspicuous over labial enamel-covered part of incisors in region anterior to fulcrum of tooth. Paradentium of incisor teeth divisible, on basis of function, into cementum-covered suspending and enamel-covered cushioning portions. Cementum-covered tooth surfaces contain embedded collagen fibers: continuous with those of peridental membrane, uniting with fibers in alveolar bone. Forces of occlusion transmitted through cementum and peridental membrane to surrounding bone. Interwoven bundles of collagen fibers intermittently placed under tension, thus allowing limited movement of tooth resulting in slight alternating negative and positive pressures upon cushioning part of paradentium. In acute scurvy new collagen-fiber formation normally accompanying continuous eruption of tooth fails to occur. When sus-

pending part of paradentium unable to withstand occlusal forces, these transmitted directly to cushioning part of paradentium resulting in alveolar atrophy described.

—*Copy*

71—Badeff, T.: **Contribution a l'étude du metabolisme général dans les paradentoses. La réserve alcaline.** Contribution to the study of general metabolism in periodontoclasia, in: Schwz. Msch. Zhkde. 47(9):901. 1937.

Badeff examined 51 paradentosis cases, in no one of which was a tendency to acidosis observed. On the contrary, the dystrophic cases of paradentosis leaned rather to alkalosis.

In the metabolism research, the paradentotics were divided into three groups: dystrophic, inflammatory, and mixed.

The dystrophic type is found among males in a marked majority of cases. (29) 25 cases showed a normal CO_2 blood content.

The mixed type numbered 17 cases; 16 normal in blood alkaline content, one showed increase.

Of the inflammatory type (5) all showed a normal CO_2 blood content, which is the measure for the alkaline reserve. Out of 29 dystrophic cases, 26 were men, while among both the other types, the sexes were equally divided.

—*H. A. R.*

72—Editorial: **Salivary antiseptic,** in: J. A. M. A. 108(15):1264. Apr. 10, 1937.

Certain German workers have found that the human saliva contains agents which not only inhibit the multiplication of many species of virulent bacteria *in vitro* but will even sometimes kill them and when this does not take place it frequently will transform them into avirulent types. It has not yet been determined whether the inhibiting substances or principles are salivary enzymes, epithelial products or antagonistic factors formed or secreted by normal oral flora. The immunizing power of saliva presents many interesting clinical problems worthy of study.

73—Goldstein, Harold, D.D.S.: **Systemic and blood picture findings in several hundred periclasia-free and periclasia-involved individuals,** (Abstr. 50), in: J. D. Res. 16(4):320. Aug., 1937.

Series of 200 *periclasia-free* persons, aged 35 to 70, recorded for systemic diagnosis, blood pressure, hemoglobin, erythrocyte and leukocyte counts, and occupation. Same records made for 200 *periclasia-involved* cases, aged 30 to 70. Reports obtained from regular medical histories. *Findings:* (*a*) Diseases, such diabetes, lues, gastro-intestinal ulcer and cancer, more frequent in periclasia-involved than in periclasia-free individuals. (*b*) Arthritis and cholecystitis more frequent in periclasia-free group. (*c*) Leukocyte counts (8,000 or over): higher in periclasia-involved group. (*d*) Hemoglobin and erythrocyte values generally same for both groups. (*e*) Blood pressure: greater number of periclasia-involved persons in lower blood-pressure brackets; greater number of periclasia-free persons in higher brackets. (*f*) Occupation: much lower incidence of peridental disease in persons engaged in "better occupations." (To be published in *J. Den. Res.*)

—*Copy*

74—Gordon, Samuel M., Ph. D.: **Action of sodium metaphosphate on tricalcium phosphate, dicalcium phosphate, calcium carbonate, and human enamel,** (Abstr. 104), in: J. D. Res. 16(4): 345. Aug., 1937.

Sodium tetrametaphosphate important ingredient in highly advertised tooth-paste and tooth-powder. Sodium metaphosphates form complex ions which, acting upon insoluable Ca compounds, including human enamel, dissolve Ca. Compounds listed in above title subjected to action of tooth-paste and tooth-powder containing stated compound. Dissolved Ca determined by modification of Clark and Collip method. Amounts of Ca dissolved, from each compound, after correction for blanks, summarized below:

	Enamel	*Tri-calcium phosphate*	*Precip-itated chalk*	*Calcite*	*Dicalcium phosphate*
Tooth-powder	13.12	14.88	25.62	26.67	26.99
Tooth-paste	9.03	5.24	28.35	19.89	20.53

Normal blood-serum contains approximately 10 mgms. of Ca per 100 cc. Experiments, still in preliminary stage, indicate action on whole human teeth parallels that on powdered human enamel. [*Author's note in proof reading:* Composition of indicated dentifrices lately modified by addition of tricalcium phosphate; decalcifying action of sodium tetrametaphosphate probably thus prevented.]

—*Copy*

75—Hatton, Edward H., B.L., M.D.: Biological and mechanical significance of epithelial attachment, (Abstr. 52), in: J. D. Res. **16**(4):321. Aug., 1937.

Thin sections of unfixed and undecalcified teeth and investing tissues of young dogs, just after eruption of permanent teeth, obtained by grinding small blocks, frozen with dry ice on small microtome. Gingiva not mechanically attached to enamel surface—may be pushed from enamel with delicate, light wire without appreciable force, but returns promptly to position immediately adjacent to enamel. Such dyes as gentian violet and methylene blue—such fluids as India ink—do not penetrate layers between gums and enamel excepting as they diffuse through soft tissues. Enamel surfaces covered by gingival tissues do not decay. Even dentin overlaid by hyperplastic pulp hard and eburnated (arrested caries). *Although mechanically epithelial attachment not important, biologically gingival tissue closely approximated to enamel very significant.*

(Italics not in original.)

—Copy

76—Karshan, Maxwell, Ph.D. and Theodor Rosebury, D.D.S.: Biochemical characteristics of saliva of Kuskokwin Eskimos correlated with dental caries and salivary calculus, (Abstr. 23), in: J. D. Res. **16**(4):307. Aug., 1937.

Paraffin-stimulated salivas from 49 of 106 Eskimos (abstracts 21-22): 17 *caries*, 28 *caries-free*, 4 *doubtful*, preserved with $HgCl_2$ (on basis of preliminary tests), analyzed for total Ca, inorganic P. CO_2-capacity, and change in Ca and P after shaking with commercial tricalcium phosphate (*J. Den. Res.*, 15: 383, 1936). Since commercial tricalcium phosphate not uniform in composition, single sample, same as in previous study, used throughout. *Results: Lower averages* in *caries* group for Ca, P. CO_2-capacity, and Ca removed after shaking; *higher* average in *caries* group for P removed after shaking. Differences between caries and caries-free groups generally like those previously reported, further indicating similarity of Eskimo caries to caries elsewhere. Same data correlated with salivary calculus: 28 *low-calculus* cases (including 16 of 17 *caries* cases) gave average values similar to caries group; 20 *moderate* or *high-calculus* cases, similar to *caries-free* group. Data suggest inverse relationship between caries and salivary calculus. Results for total Ca and inorganic P similar to those reported by Tenenbaum and Karshan (abstract 20). Attempt to coördinate chemical data with bacteriological data for Eskimos (abstract 22) shows little relationship in individual subjects. Two methods may yield information on dissimilar mechanisms, both related to activity of caries.

—Copy

77—Kronfeld R., D.D.S., M.D.: Histologic analysis of child's jaws in malocclusion, in: J. D. Res. **16**(4): 319. Aug., 1937. Abstr. G. R. S.

Specimen: upper and lower jaws of child of 11. Upper right anterior teeth markedly crowded; upper right cuspid forced to erupt on labial side of arch. Anterior portion of upper jaw sectioned in horizontal plane, to facilitate study of position of roots of anterior teeth. On left side roots in normal alignment; bone and periodontal membrane normal. On right (crowded) side, space between central incisor and first bicuspid greatly reduced; apex of lateral incisor located on lingual side of root of cuspid, latter forced distally, as indicated by arrangement of bone on mesial side of root. Periodontal membrane on distal side of cuspid: very unusual enlargement and increase in number of blood vessels—tissue reaction described by Oppenheim as characteristic of periodontal membrane on pressure side of human teeth subjected to orthodontic treatment. (To be published in Angle Orthodontist.)

—Copy

78—Labbé, Gonzalés - - - - : Etude de la phosphatase de l'os sain et de l'os altéré by polyalvéolyse. Study of phosphatase found in sound bone and in bone altered by polyalveolysis, in: Revue de Stomatologie, **39**: 1937.

In this work the author asks: What bond of union exists between pyorrhetic alveolar atrophy and phosphatase?

Phosphatase is a ferment discovered by Robison in 1923, whose action is important in calcification and whose origin is in the blood globules.

From fresh cadavers, portions of bone were dissected from the tibia and from the diseased maxilla and the respective quantities of the ferment obtained were compared. Contrary to the expected finding, phosphatase was from two to five times more abundant in the pyorrhetic bone than in sound bone. The explanation of this phenomenon

remains uncertain. The author makes some hypotheses as to pathogeny, while confessing the impossibility of verification.

It seems best to assume that the increase of ferment in the case of the pyorrhetic bone is a defensive mechanism intended to hinder the resorption of the alveolar bone while augmenting the possibilities of calcification of the damaged portions.

—*H. A. R.*

79—Osborn, T. W. B., M.Sc., J. N. Noriskin, and J. Staz (Johannesburg, South Africa): **Comparison of crude and refined sugar and cereals in ability to produce in vitro decalcification of teeth.** (Abstr. 109), in: J. D. Res. 16(4):347. Aug., 1937.

Decalcifying power of crude and refined sugars and cereals tested, on sections of teeth, in mixtures of sugar and saliva or cereals and saliva. Decalcification determined by two independent visual examinations; also with sharp probe and by staining with Grenacher borax carmine. In white-sugar mixtures, high percentage of teeth decalcified; in crude-sugar mixture, very few. With whole-mealie meal, very few teeth decalcified; with highly refined flour mixtures, significantly greater proportion. Results indicate presence of "protective agent" in crude sugar and cereals: removed in processes of refining; independent of prevention or reduction of acid fermentation. (Published in *J. Den. Res., 16:* 165, 1937.)

—*Copy*

80—Siegmund, Herbert: **Die augenblickliche Situation der wissenschaftlichen Paradentoseforschung.** The present status of scientific research on paradentosis. in: *Dtsch. Zahn-Mund- und Kieferheilkunde* 4:81-89. Feb., 1937.

The tooth and the paradentium form a functional unit. The morphologic structure of the paradentium depends entirely on its function which is determined by the occlusion and articulation on the tooth. Intensity, direction, and length of time of the active forces are important factors in the anatomy of the paradentium. Upon pressure, resorption of the bone occurs, upon traction, apposition of the bone. The same changes in the bone structure occur in malocclusion as in orthodontia where artificial forces are applied. When functional equilibrium is established the changes in bone cease. Loss of functional equilibrium is an important mechanical factor in causing paradentosis. Occlusal wear and continuous eruption of the tooth (Gottlieb) are important in this connection. Active eruption is the condition when alveolar process and new attachment grow to keep pace with the new position of the tooth, passive eruption when the epithelial line is lowered together with recession of the alveolar border. The author believes the function of the tooth determines whether the continuous eruption of the tooth during the lifetime shall be active or passive. This explains the great differences in alveolar level between several teeth in the same dentition. As wear of the contact points occur the teeth drift mesially to take up the space. This is accompanied with bone resorption on the mesial sides of the roots and apposition of alveolar bone distal to the roots. If the migration is slow enough and occurs only late in life, then the tooth has a long life. If the passive eruption occurs early in life then symptoms of paradentosis will appear and early loss of the tooth is possible. New layers of cementum on the root tend to compensate for passive eruption. Such new layers of cementum are more cancellous than early formed cementum due to metabolic changes in the pericementum. Another factor tending to compensate for the increased length of clinical crown in relation to clinical root is occlusal wear of the tooth. If normal wear occurs in a perfect occlusion, a long life for the tooth is possible. If the grinding capacity is disturbed as in malocclusion or malarticulation, paradentosis may occur because of uncoördinated forces acting on the tooth to cause resorption of bone. The basis for paradentosis may exist at the time the permanent teeth first come into occlusion.

Häupl—Lang point out that mechanical forces alter the metabolism of bone, dentin, and cementum as well as the pericementum. The metabolism of the protoplasm of these tissues as well as the calcification processes are affected. The author believes that organic cells are decomposed by undue pressure leading to localized acidosis decreased oxygen tension, lowered metabolism and deposits of metabolic debris. Absorption of bone and inflammatory reaction result. (The author presents a detailed study of the effects of mechanical pressure and traction on the alveolar walls which is similar to that of Lundquist. *J. A. D. A.* 24:1577-85—

Sept. 1937 which see F. R.) The author does not agree with Pommer who claims that resorption of bone is caused by increased blood pressure in the tissues.

Other important characteristics of paradentosis are inflammation of the gingiva, pocket formation, and bleeding. If bacteria and putrefactive products are absorbed through the gingival epithelial layer into the subepithelial tissues necrosis of the pericementum occurs. This causes progressive inflammation, solution of the pericementum and alveolar bone, the formation of granulation tissue and proliferation of the epithelium in the effort to cover the ulcerated surface. Inflammation is the tissue reaction to an irritant. It does not determine either the nature and cause or course and prognosis of the damage.

There are several internal as well as external factors that influence bone resorption in paradentosis. Age is one of them. It is well recognized that nearly everyone will lose his teeth from paradentosis at advanced age if not before. How early or how late will depend partly on constitutional factors. Of these factors the endocrine organs which govern metabolism are important. Increase or decrease of pituitary function leads to crowding of the teeth or changing their positions. Acromegaly often causes separation of the teeth followed by paradentosis. In cases of the "Cushing's Disease" (pituitary basophilism) paradentosis occurs at an early age, possibly due to a disharmony of the hormones. Severe paradentosis may or may not be found in disturbances of the thyroid secretion.

81—Tenenbaum, Benjamin, B. S., D.D.S. and Maxwell Karshan, A. M.Ph.D.: **Factors in saliva correlated with occurence of salivary calculus,** (Abstr. 20), in: J. D. Res. **16**(4): 305. Aug., 1937.

Saliva of persons with and without calculus analyzed for total Ca, inorganic P, pH, protein. Saliva, stimulated by chewing paraffin, collected in three successive portions, 1-3 hours after breakfast. First portion (1-2cc.), collected under paraffin oil, used for initial pH; second portion (10-12 cc.) used for Ca, P, protein; third portion (1 to 2 cc.), collected under oil, used for second pH determination. Of 49 persons 20-52 years old, 29 were calculus-free; no previous dental prophylactic treatment for at least one year. Remainder, varying degrees of deposit. Mean values for Ca and P in calculus-free group lower than in calculus group: difference in mean of Ca definitely significant; difference in P probably significant. Mean pH lower, and mean protein content higher, in calculus-free than in calculus group; but neither difference statistically significant. Data indicate Ca and probably P in stimulated saliva correlated with occurrence of calculus. (Aided by grant from Carnegie Corporation.)

—Copy

82—Teuscher, George W. and Leonard S. Fosdick: **Retention of reducing sugars in saliva after mastication of carbohydrates,** (Abstr. 123), in: J. D. Res. **16**(4): 354. Aug., 1937.

Number of bacteria and retained amount of carbohydrate, in mouth, possible factors in caries. Semiquantitative determination (Benedict solution) of sugar retention, after meals and ingestion of candy (85 boys): gradual disappearance of sugar from saliva after consumption of various sugar-containing foods, rate of removal varying with individuals—those more susceptible to dental caries retained sugar longer. Effect, on mixed saliva, of ingestion of sugary desserts and candy: "larger percentage of sugar remained longer time."

—Copy

(d) Clinical Etiology

(Continued from 8(2): 115. July, 1937)

364—Abdala, Dr. Salvador: **Absorcion alveolar de origen especifica, (Caso clinico).** Alveolar Absorption of syphilitic origin. Revista Estomatologica de Cuba. June, 1937.

Sra. X, 35 yrs., tertiary lues, complete aphonia owing to a laryngeal inflammation. On account of trismus, unable to open mouth, thus rendering intra-oral examination impossible. Complained of toothache due to a vestibular phlegmasia extending from the second left upper premolar to the second molar of the same side. External palpation produced pain. Gingivae apparently normal. Light salivation. X-ray showed marked alveolar absorption.

—H. A. R.

365—Ayllom, Pedro: **Le probléme étiopathogénique de paradentose.** Etio-

pathogenic problem of paradentosis. La Odontologia. Aug.-Sept., 1936.

A very detailed study of this disease has been made as to pathogeny, evolution, constitutional influence of the soil, effects of local factors, interrelation between local and general states; relations of the endocrine system, pancreatic disturbances, hepatic insufficiencies, general osseous dystrophies, etc., to the paradentium.

The author's conclusions are as follows:

1—The accountability of the soil is recognized.

2—Stomatologic observations are not enough for the special forms of disease in question. Both local and general examinations of the patient needed.

3—Special service of stomatology is necessary to reduce observations to formulae, to record statistical data, etc.

4—Important to establish special commissions to investigate paradentosis.

5—Special courses in the schools of odontology are needed.

6—The scientific evolution of the etiologic and pathologic problems of paradentosis is desirable. This disease is a social scourge.

—*H. A. R.*

366—Badanes, Bernard B., D.D.S. and Clara B. Parodneck, M.S.: **The influence of emotional states upon tartar accretion.** J. A. D. A., **24**: 1421. Sept., 1937.

The concept of health should be enlarged to include the oral cavity. Psychic and somatic reactions are so interrelated that they must be studied together. Mental poise is an expression of health just as health is expressed in certain types of behavior. Psychoneuroses and intense emotions, by affecting adversely the hormonal secretions of the endocrine glands are bound to give rise to a variety of constitutional disorders.

"Neurasthenia is one of the many ailments whose role has been substantiated in its etiologic relation to calcium concretions in both renal and salivary calculi, originating from the disturbance in the phosphorus-calcium balance." This disturbance may be due to avitaminosis and bacterial activity as shown by Osborne and Mendel. When the resistance to bacteria is lowered, these organisms may produce ammonia and calcium phosphate will precipitate in an alkaline medium. Excess of calcium in relation to phosphorus may result in both urinary and salivary calculosis. Calciuria and oxaluria bear a direct relation to the formation of much tartar about the teeth.

Black's observation that an intensification of salivary deposit occurred from one to three hours after a heavy meal, which corresponded to the post-prandial tide, is worth noting. Alkaline tides resulting from psychopathic conditions may last for days or months.

Calcareous deposition results from a shortage of the proper acids needed to combine with the basic elements. . . . "It seems to be excessive calcium relative to phosphoric acid which leads to precipitation . . ." (Young).

—*G. R. S.*

367—Barnes, H. N. V.: **A dental examination of the inhabitants of Tristan Da Cunha.** J. A. D. A., **24**:1566. Sept., 1938. (Abstr.)

The low incidence of dental disease found in 162 inhabitants of Tristan da Cunha in 1932 prompted a second examination of the whole population of 183 persons. It was found that the percentage of carious teeth had increased to 4.2 per cent as compared with 1.84 per cent in the examination five years previously. Gingivitis had increased from 3.21 to 26.20 per cent; pyorrhea from 1.22 to 4.30 per cent, and 50.3 per cent of the mouths were free from caries and extractions as compared with 83.33 per cent in 1932. The habits and diet of the community had not changed except for an increase in the consumption of sugar and flour and the introduction and use of toothbrushes. Two tables compare the observations made in 1932 and in 1937.—*Brit. D. J.*, 43:86, 1937.

—*B. G. Bibby.*
—*Copy*

368—Blessing, Georg: **Wesen und Bekampfung der Paradentose.** Nature of paradentosis and struggle against it. D. Z.-M., zu Mund- und Kieferheilkunde. **4**(6):418. 1937.

Blessing gives the usual brief survey of opinions on paradentosis and the changes in methods of treatment during the past twenty years. He describes the rôle played by local and constitutional stimuli, noting that the deepening of the pockets is due primarily to infection (streptococcic, spirochetic, or fusiform bacillary). He then discusses the significance of mal- or traumatic occlusion, to the rôle of constitutional disease, to

hereditary malocclusion, to undernutrition on the one hand; and on the other, to misuse of alcohol, tobacco, etc. Inner secretory or nervous disturbances also favor the outbreak and development of periodontal disease. The rôle of diet is touched upon; the cause of frequency of relapse is explained. Article ends with a discussion of public health relations to paradentosis problem, and racial hygiene.

—*Transl. of Abstr.*
—*H. A. R.*

369—Boyle, P. E., O.A. Bessey and S. B. Wolbach: **Experimental alveolar bone atrophy produced by ascorbutic acid deficiency and its relation to pyorrhea alveolaris.** J. A. D. A., 24: 1568. Sept., 1937. (Abstr.)

Changes in the peridental tissues due to vitamin C deficiency with resultant failure of intercellular substance to form properly were investigated. The pyorrhetic, scorbutic and ascorbutic acid-inadequate diet, alveolar process and peridental tissues were identical. Roentgen-ray and histologic study revealed rarefaction of the alveolar bone and widening of the peridental membrane, with ultimate loosening of the teeth and atrophy of the bone in acute scurvy. Similar changes take place more slowly in animals at low ascorbutic levels. The bone rarefaction is a result of osteoblastic failure, and loosening or drifting of the teeth expresses a weakness of the supporting bone and the suspending fibers.—*Proc. Soc. Exper. Med. & Biol.*, 36: 733, June, 1937.

—*Hamilton Robinson.*
—*Copy.*

370—Chatellier, Jean: **Considérations sur la vie et la mort des fondations ligamento-alvéolaires. Étude panoramique en trois époques.** Considerations on the life and death of the alveolodental ligaments. Panoramic study of three epochs. Revue de Stomat. 39(3):194. Mar., 1937.

1. The first epoch consists of a dynamic period, reparative at the beginning, evolving during infancy and characterized by the existence of traumatism of the articulation, congenital and latent. Its goal is the victorious resistance of the ligamento-alveolar articulations and their reparative quality. During this period the physiologic equilibrium is complete.

2. A static period slightly reparative during the adult age. Traumatisms of the articulation equalize themselves with the resistance of foundations, save in those regions where the teeth are much exposed and much traumatized. Such teeth present monoalveolysis. Physiologic equilibrium is only partial.

3. A period of dislocation (disarrangement?) develops between maturity and senility. It is accompanied by the breaking down of the alveolar region and the teeth supported thereby, under the action of general causes and traumatisms. During this terminal period the physiological equilibrium gives place to mathematical disequilibrium and the syndrome of Fauchard develops.

Even if from the many states called pyorrhetic, a specific malady should emerge in the future it may be easily adapted to the new theory.

It has seemed to the authors a matter of interest to make their study on the life and death of the ligamentum alveolaris foundation as a whole; for only in this way can the evolution of this region be understood. The panoramic view apparently corresponds with the reality in the majority of cases.

BEGINNING PERIOD	STATIC PERIOD—MONOALVEOLYTIC	DEBACLE-POLYALVEOLYSIS
Local causes without local consequences, thanks to general condition. Children	Local causes, traumatic, especially Adult Age	General causes and secondary infection Maturity and Old Age
Dynamic Period: Physiologic equilibrium, perfect. Cure	Static Period: Physiologic equilibrium, partial Treatment of value.	Disruption: Breaking down. Physiologic equilibrium, dislocated. Extraction-exfoliation

—*Transl.*
—*H. A. R.*

371—Davidoff, ——— Dr. med. and Dr. med. Dent. (Leipzig): **Die Paradentosis mit speziellen Berücksichtigung an Störungen des Verdanungstraktus als ätiologische Momente: und klinische und experimentelle Untersuchungen einer rationellen organ-spezifischen Therapie bei derselben.** Paradentosis with special reference to disturbances of the digestive tract as etiologic stimuli; and clinical and experimental researches in rational and specific organ therapy. Zahnärztliche Rundschau, **46**(3)17; (4)146. 1937.

Capillary microscopy has for some time offered proof that in every inflammation there are to be seen in addition to an altered marginal periodontium, morphologic changes of the capillaries. The present author is persuaded that disturbances of the alimentary tract are of the greatest importance not only in the etiology of the paradentoses but also for their influence on therapy, which appears in a new light. This has led to the opinion that most of the paradentoses must be regarded as paradental manifestations of former or still present organic or functional changes in the digestive tract.

These researches included two groups of patients: 1) already reported in 1935 embraced those patients with primary paradentosis, while the patients were still clinically sound: 2) included patients in whom a manifest or latent gastro-intestinal or some similar general affection existed, accompanied by paradentosis. In perhaps fifty persons from both groups studies were instituted as to the Ca and P values in blood and serum. The findings were "that there is scarcely a case of periodontoclasia in which there does not exist either a gastro-intestinal disturbance or some similar general ailment, past or present."

The author describes a number of cases in support of his contention.

—*H. A. R.*

372—Holst and Jarlow: **Klinische Untersuchungen ueber die Beziehung zwischen medizinischen Allgemeinleiden und Paradentose.** Clinical researches on the relation between general disease and periodontoclasia. Tandlaegebl. (Dän) **10**. 1937.

Authors conclude as follows:

The evident connection between paradentosis (periodontoclasia) and internal disease which is stressed by certain authors is not confirmed by any evidence in their material, and in their studies cannot be found so consistently, since even in the limited study of one hundred paradentotic cases it should have appeared. They have observed no striking occurrence of syphilis, tuberculosis, endocrine, or other metabolic disturbance, in cases of paradentosis.

373—Iskraut, H.: **Zum Problem der Parodontose.** Concerning periodontoclasia. Deutsche Zahnärztliche Wochenschrift, **40**(1): 9. 1937.

Dr. Iskraut thinks that under no circumstances can purely local causes be assigned as the etiology of this disease. Much more often there are disturbances of the activities of the growth hormones, especially the hypophysis and thyroid gland secretions. The influence of the growth hormones of the adrenals is little known; its behavior is bound up with that of the other internal glands, while the hypophysis "takes and steers man's wheel."

A thyrotropic hormone of the anterior portion of the hypophysis has been successfully demonstrated. It is now evident how the growth hormone functions; and in case of hypofunction paradentoses appear, consisting in disturbances of the process of apposition in the bones.

Despite this, the hormonal system of therapy presents marked difficulties.

—*H. A. R.*

374—Linka, —— (Trieste): **Beitrag zur Pathogenese u. Therapie der Paradentose.** Contribution to the pathogenesis and treatment of periodontoclasia. Paradentium, **9**(1):1937; Z. Rdsch. **46**(6): Feb. 7, 1937.

This article is limited to the presentation of new observations, and the question: Which are the distinguishing substances in the different metabolic disturbances which lead to periodontoclasia, as for instance, diabetes, avitaminoses, endocrine peculiarities, etc.?

The conclusion is that the organism selects and eliminates harmful materials, such as arsenic, bismuth, etc., through the gingival margin availing itself of the leucocytes as carriers. This process may run its course unobserved, when the excreted material is present only in limited quantity. But if this material, formed in the body itself eventually increases in mass, the continued stream of leucocytes cannot leave the gingival margin wholly unharmed. Since this

excreted material must pass over the tooth cervix, it is clear that given a certain intensity, a loosening of the gum from the tooth takes place. Pockets are formed in which the leucocyte stream settles and becomes perceptible. If this condition lasts long, numerous leucocytes die in the collection of pus in the pockets; and out of the nucleoli, the nuclei form uric acid which attacks the alveolar margin and causes it to disappear. We are dealing with a sort of gout, complicated by infection of the contents of the pockets. Not only this, but the leucocytes themselves are sedimented into the alveolar process as osteoclasts and may cause cavities, falsely interpreted as abscesses. In this way the alveolar "gaps" may be explained where general pyorrhea has not yet developed. Microphotographs are shown where the tissue at the gingival margins is so crowded with leucocytes as to render the structure unrecognizable.

As regards treatment, massage mechanically disperses the leucocytes; and the operative opening up of the pockets opens up a path of egress. But in the nature of the disease, a complete healing cannot be expected unless the metabolic disturbance at its source can be overcome.

—*H. A. R.*

375—Mahé, Gustav: **Rélations entre la carie et les autres altérations du systéme dentaire.** Revue de Stomat., **39**(7):535-543. July, 1937.

Though caries is the most common and fatal of the maladies attacking the teeth, even its problem might be solved by study of these diseases together not singly. For this article the author selected three of these dental diseases of which paradentosis is most considered in this abstract.

P. Dubois (Aide-Mémoire du Chirurgien —Dentiste, 188) states that the teeth attacked by "expulsive periodontitis" are almost exclusively "hypercalcified," which is to say immune to caries. This statement appears simple and so to speak, absolute; but, at that time, the diagnosis of expulsive periodontitis was much less comprehensive than that of today into which, beside all the alveolyses, many other things enter, even lesions due to tooth abrasion, ... For the time is perhaps not wholly past when it was considered "good form" among patients to have "pyorrhea."

Author quotes Dr. Lester R. Cahn (1932) who says:

> It is a common observation that teeth of this type (pyorrhetic) are free from caries . . . I have examined the pulps of many pyorrhetic teeth and found that the outstanding lesion is interstitial calcification, often strongly advanced, which is a decided evidence of a sluggish circulation . . . This tissue (the pulp) becomes the seat of circulatory retardation . . . and although the pulp retains enough vitality to function as a nutritive organ, it no longer has an influence on the calcification of the tooth. In consequence of the decreased blood supply the liability of salt abstraction, with resultant caries is greatly diminished.

Here we meet again the conception of Leriche as to osteogenesis by slowing the circulation, and the oscillating role of the odontoblastic layer, subjected to two circulations; the one "afferent" bringing the calcic materials, the other "efferent,"—capable of carrying them away. But this conception enlarges the problem, and caries ceases to be a purely local affection of entirely external origin. P. Dubois committed an error inevitable at that time of attributing hypercalcification to a functional superactivity of the pulp; Cahn on the contrary believes that periodontoclasia is the cause of the pulpal degenerescence. The author believes the truth to be that the two changes are the simultaneous consequences of a one and only cause.

This is in fact the endocrino-sympathetic complex already encountered in the etiology of caries as a link between the general biologic disequilibrium and the local distribution of the odontoblastic layer . . .

The author concludes that for these reasons the two diseases, caries and regressive alveolysis (or paradentosis) may be considered as closely related by a common mechanism producing one or the other according to how it is used; and that for these reasons, study of the two affections together and not separately seems to offer a possibility of clearing up one by the other.

—*H. A. R.*

376—Marichal, B. (Tegucigalpa, Honduras): **Considerations on Paradentosis. Osseous alveolar atrophy: its causes.** Revista stomatologica de Cuba, Aug., 1937.

In paradentosis, inflammation of the gums, the pyorrheal pockets and the suppuration are not essential without secondary complications of the affection. The author believes that the destruction and osseous atrophy are more important. Metabolism is governed

directly by the secretion of the thyroid, because it contains bone; and the organic ectoderm is profoundly influenced by the thyroid secretion. Hence, a good or faulty dentition indicates a perfect or imperfect thyroid function.

In paradentosis general predisposing causes may be diabetes, diathesis and others. A large number of diabetics (pancreatic insufficiency, etc.) show signs of hyperthyroidism. Sugar is frequently noted in hyperthyroidism.

In hyperthyroidism, the equilibrium is altered in the sense of increased acidosis. Following a disturbance of Ca metabolism, an intense decalcification is seen, as the result of a heightened metabolic process.

1. Vaso-dilatation and the new vascularization, intensifying the circulation of the blood, provoke the lysis of the alveolar process; because the hyperactive circulation is incompatible with the maintenance of osseous density.

2. Frey and Hulin admit the theory of Leriche and Policard, which assumes that the more active the circulation in the bone, the less can the density of this tissue be maintained.

3. According to the intensity of the infection, the osteolysis is complicated with osteitis. Physical disorders become injected among these biological disturbances so that as the traumatic action organizes and develops owing to the above mentioned factors, the physico-chemical modifications peculiar to these phenomena are acquired.

4. All these morbid disturbances have repercussions on the others, thus favoring the microbic invasion which in the circumstances in point is more or less premature. Hence it results that the cellular framework is destroyed, necrobiosis follows; suppuration accompanies destruction of the bone by osteolysis; destruction of the alveolodental ligament; and as finale, the expulsion of the tooth, due to the retention of the disintegrated proteins.

377—Nielsen, ———: **Paradentosis.** Tandlaegebl. (Dän), **10.** 1937.

Author's summary:

1—From our researches we consider paradentosis (periodontoclasia) a result of gingivitis.

2—Advanced age disposes to change of gingivitis into periodontoclasia.

3—Certain internal diseases, especially diabetes mellitus appear to favor this change from gingivitis into periodontoclasia; but the etiological connection between internal disease and paradentosis is by no means so clear as has been asserted earlier.

4—In the present material (author's researches) periodontoclasia occurs in women not more frequently than in men, rather the reverse.

5—Our results indicate that the rational prophylaxis of periodontoclasia must be directed against gingivitis, especially at an early age.

378—Orban, B.: **Knorpel in periodontium.** Z. Stomat., **35**(H. 8):532. 1937.

Author points out that malocclusion can lead to points of pressure and contusions in the periosteum. Near such points may be found cartilage formations.

It is not unlikely that the change in the periosteum to hyaline cartilage ends with calcification.

—*H. A. R.*

379—Roy, M.: **Quelques notions fondamentales pour la compréhension de la pyorrhéa alvéolaris.** Some fundamental conceptions for the understanding of pyorrhea. Proc. Soc. Odont. de Paris. Rev. de Stomat., 1937.

Dr. Roy considers the following ideas the basis of our knowledge of pyorrhea alveolaris or paradentosis:

1—Alveolar resorption is the initial fundamental lesion of alveolar pyorrhea.

2—The intervention of a disturbance of the general economy acting upon the centers regulating alveolar nutrition to cause resorption is the primordial element indispensable to this basis.

3—All alveolar resorption which, without the superaddition of inflammatory resorption is not a cause to be classed as general, has no right to a place in the picture of pyorrhea.

4—All the other pathologic manifestations which accompany the evolution of pyorrhea are subordinate to the appearance of these primordial factors.

5—The precise knowledge of the respective relations of the evolution of the diverse manifestations which characterize the development of pyorrhea is the basis of

all rational therapy of this disease, whose primary requirement—as much prophylactic as curative—is energetic gingival brushing, practiced daily by the patient.

—*Transl.*
—*H. A. R.*

380—Rustishauser, E.: **Analyse de la loi de Kölliker son application aux osteodystrophies causés par les métaux, et a la paradentose humaine.** Application of the law of Koelliker to paradentosis. Annales d'anatomie pathologique 14(2)107-20. Feb., 1937.

The Law of Koelliker indicates that the osseous lesions localize by preference at the zones of growth and at bony parts most responsive to static and functional effort. We may add the thick internal layer of the periosteum. Exogenous and endogenous fibrous osteopathies are exhaustively discussed. In the acidotic osteopathis paradentoses are frequently found, also in hyperthyroidism and hyperparathyroidism. But they also occur without acidosis, in hypercalcemia, or osteitis fibrosa. It is a morbid state whose evolution progresses step by step. Its active periods alternate with those where the metabolism is normal. Minor metabolic modifications provoke only paradentitis. The rebuilding of the alveolar walls, according to this author, is impossible. The alveolar process is definitely formed dating from the time of eruption. There is no tangential force sufficiently strong to rebuild the effaced alveolus.

En resumé the chemical analysis of bone confirms the Law of Koelliker. It is the most fragile part of the jaw which is the most quickly attacked.

—*H. A. R.*

381—Siegmund ———: **Rückblicke und Ausblicke auf die wissenschaftliche Paradentoseforschung.** Tandlaegebl. (Dän) 10:479. 1937.

The best introduction to the etiology and clinical history of the paradentopathies is probably found in the study of periodontoclasia.

The alveolar pyorrhea of a past, technically empirical dentistry has become a diseased life process, the periodontoclasia concept, which has awakened the enlightened and just interest of the entire medical profession. Beyond the symptom of suppuration from the gingival pockets, and beyond the limited area of the gingivae, it concerns the pathologic anatomist, as well as the internist and the nutritionist; and reaches the endocrinologist as also the student of race and constitution.

Siegmund stresses in the beginning the current researches and conceptions in order to lay a broad foundation on which to base new ideas. Of significance especially in a practical sense are the local conditions found in the dentition. "The paradentium offers the direct point of attack of the mechanically functioning stimuli passing from the tooth; these extend over the cementum, the periosteum, the cortical investment of the alveole and the retro-alveolar *spongiosa* space to spread over the body of the jaw.

This physiologic unity is more than the sum of different kinds of tissue; it is an entity of structure, undetachable it is true, but of common function. With the tooth the adherent paradentium forms the real *organum dentale.* According to this system, the tooth is no independent individual, but a functional sharer; the paradentium is itself a part of the entire organism.

Hence it seems to Siegmund that we are no longer justified in seeking the cause of periodontoclasia in some remote disease of the organism; but that the paradentosis in itself is the expression of a constitutional and biological susceptibility, as a stigma of extreme variation."

382—Stuteville, O. H., D.D.S.: **Injuries to the teeth and supporting structures caused by various orthodontic appliances, and methods of preventing these injuries.** J.A.D.A. 24:1494. Sept., 1937.

A report of a study demonstrating the changes that take place in the teeth and supporting tissues when orthodontic force is applied to the teeth. The specimens presented are from dogs and human beings in whom the teeth were moved with measured force active for known distance.

Laboratory methods of orthodontic treatment are condemned and the importance of very special training in knowledge and skill for the orthodontist emphasized. It is highly important that a minimum of trauma to the teeth and the periodontal tissues be produced by orthodontic interference. In every orthodontic case treated by mechanical appliances there are certain tissue changes produced that are pathologic, and the slight in-

juries to the teeth and supporting tissue is often justified because they are the lesser of two evils.

Possible injuries to the peridental membrane may be asphyxia of the tissue caused by squeezing it between the surface of the tooth and the alveolar bone, which if continued long enough will result in necrosis, and a sudden heavy force causing tearing of the membrane's fibers on the side of tension. By the use of certain types of springs and where there is interference to the movement of the teeth the fibers may be torn and crushed on both sides of the roots resulting in loosening of the teeth. There may be injuries to the surfaces of the roots, to the alveolar bone, (even to the elimination of the labial or buccal wall) to the gingivae, and to the pulps. It is safe to say that root resorption is produced in 99% plus, of all cases, and necrotic areas in the peridental membrane in a large percentage of them.

The experiments showed that many of these injuries are repaired and healed but it is easy to over step the bounds of safety. Rigid appliances seem to cause less root resorption. Emphasis is placed upon the great importance of allowing sufficient time to elapse between adjustments for the supporting tisues to return to normal.

—*G. R. S*

383—Thielemann, K. (Frankfort a/M): **Equilibrium of the articulation.** Korresp.-Bl. Zahnärzte. **61**(4):120. 1937.

The establishment of an equilibrium of the articulation is the most workable means in dental prosthetics toward the re-establishment of the biologic equilibrium of the teeth and periodontium. The goal of the dentist is the systematic maintenance of the equilibrium of the articulation, the most effectual means in the dental prophylaxis of paradentosis.

On social grounds also, this measure should find the widest spread, since compared with other measures, this calls for only a trifling outlay.

384—Van Vouno, N. C.: **Paradentose und allergischen Mundabweichungen.** Periodontoclasia and allergic oral derangements. Tijdschr. Tandhkde, **44**(10):853. 1937.

The author, a dermatologist, advocates closer coöperation between dentist and internist for the accomplishment of therapeutic ends.

Remarks follow on the allergic influence in general and on the possibility of an allergic significance of the bacteria in carious cavities for the etiology of periodontoclasia.

(e) Symptomatology and Diagnosis

(Continued from 8(2):122, July, 1937)

279—Aksoy, Z. C. B. (Istanbul): **Sur la valeur de la réserve alcaline du sang dans le diagnostic de la pyorrhée.** The value of the alkaline reserve in the diagnosis of pyorrhea. L'Odontologie **75**(9):532. 1937.

According to statistics made from persons with symptoms resembling chronic rheumatism and made by many observers in various Turkish schools in which Aksoy has served, he has observed that girls and boys, starting from the condition of hormonal disequilibrium at the age of puberty, undergo a prepyorrheic stage. From statistics of hundreds of students A. finds that this stage manifests itself chiefly among girls; some of these may be diabetic, but the greater number suffer from anemia or hepatic dysfunction. Even the other general causes, heretofore considered classical, can play a primary (M. Roy) role in the alveolysis which constitutes the initial cause of pyorrhea. There may be suppuration due exclusively to local causes in the pyorrheal culs-de-sac; but these pyorrhagias disappear completely with the suppression of the local causes.

The author's main point is the part played by the elevation of the alkaline reserve of the blood while the osteolysis is slight. In his opinion, in a genuine periodontoclasia, a perfect prophylaxis is necessary—removal of all tartar, a polishing equally painstaking;—but it is also necessary to determine the alkaline reserve of the blood.

Résumé:—In order to know whether or not a patient is suffering from a true periodontoclasia, that is to say whether or not the alveolysis is continuing, it is necessary to know the alkaline reserve of the blood. To be enabled to give a sound prognosis, this determination should never be omitted.

Also it must be ascertained whether or not hepatic insufficiency is present. If the alkaline reserve is normal, the treatment aimed at suppression of the pockets will be

efficacious and lasting; on the contrary, where the alkaline reserve and liver functions are not normal, results obtained by local or surgical therapy will be always temporary. Although the degree of hormonal equilibrium may be discovered by other examinations, the most practical determination is that of the alkaline reserve of the blood.

—*H. A. R.*

280—Bert, René: **Sur la tuberculeuse gingivale.** On gingival tuberculosis. A review of three clinical cases. Rev. dental de Chile **29**(9-10). Sept.-Oct., 1937.

The author reviews our knowledge of buccal tuberculosis. With Hans Moral, he admits the occurrence of a form, primitive, but rare, and a secondary form, supervening in the course of pulmonary or intestinal tuberculosis.

Clinically he points out three varieties; lupus, verrucous and ulcerative; the last is the most frequent of tubercular manifestations on the gingivae. Symptoms, evolution, diagnosis and treatment very thoroughly described.

281—Deliberos, J.: **Le role des affections gingivo-dentaire de la cutané pathologie.** The role of gingivo-dental affections in pathology of the skin. Thése de Paris. 1937.

These gingivo-dental lesions act directly or indirectly on the skin, in form of (1) microbic dermatoses; (2) remote infections —reflex reactions. The study is divided somewhat as follows:

a—clincal and bacteriological study of the lesions susceptible to pathogenic action on the organism at a distance.

b—cutaneous repercussions of gingivo-dental irritations.

c—microbic dermatoses in relation to contiguity or proximity of gingivo-dental infection.

d—dermatoses in relation to infective gingivo-dental foci by the blood route.

It is more difficult to express the direct skin repercussions. Only modification or amelioration of the dermatosis by treatment of the gingivo-dental lesions can give the answer. Of all the theories, that of the role of infective dental foci seems most important, and gives a satisfactory interpretation of the various forms.

—*H. A. R.*

282—Editorial: **Oral and systemic disorders.** J. A. M. A. **109**(3):211. July 17, 1937.

Editorial discusses connection between oral and systemic disorders. It is important to recognize and correlate symptoms. Flourine discoloration and the blue line of lead, bismuth, mercury and other metallic poisons are well known. Vincent's infection is often associated with granulocytopenia, and subacute scurvy can sometimes be identified by oral symptoms alone. It has never been adequately proved that there is a definite relationship between tuberculosis and the incidence of lesions of either hard or soft structures of the mouth. No excess of dental or periodontal diseases was found in an extensive study of tuberculous patients at Montefiore Hospital and even in advanced cases with extreme debilitation there was no increase in gingival erosion, caries, or in alveolar destruction or tooth de-mineralization. Secondary lesions such as ulcers of tongue or buccal mucosa were unusual and Vincent's infection not more frequent in the tuberculous than in others. Increased caries and erosion in some tuberculosis cases may be attributed to constant holding in the mouth of lozenges with high sugar content for the purpose of relieving dryness of mouth and throat and preventing coughing.

283—Le Norcy ———: **Periodontoclasia and the glands of internal secretion.** La Rev. Odontolog., **59**(3). 1937.

Le Norcy has established the fact that in many cases of periodontoclasia, disturbances in the hepatic function are observed. He concludes that periodontoclasia is the expression of a neuro-endocrine disturbance. Neither the vegetative nervous system nor the mutual interplay of the glands of internal secretion can normally be maintained if the body fluids are loaded with harmful substances or deficient in useful ones. If the material from the exterior, nourishment, vitamines, etc., after passing through the portal veins, taking up hormones on the way over the hepatic arteries, finds its pathway through the liver closed to it, the activities of the various organs cannot be rightly exerted. The liver holds the toxic materials, makes selection among them, assimilates and changes, or ejects and discards them.

Briefly, in a large number of disturbances of the vegetative system, or of the irregular functioning of the glands, we find, it is true, no periodontoclasia; for not every hepatic

disturbance leads to periodontoclasia. But, in an overwhelming majority of the cases of periodontoclasia, an insufficiency of the liver or actual hepatic dysfunction was found.

—*H. A. R.*

284—Martin, David J., B.D.S., D.D.Sc.: **The blood associated with pyorrhea alveolaris.** D. J. Australia 9:488-93. Aug. 2, 1937.

Blood examinations were made on a number of patients in an endeavor to ascertain whether pyorrhea was secondary to some disturbance elsewhere in the body which would operate in the nature of a predisposing cause. Blood counts were taken before and after the treatments for pyorrhea and if the conditions were entirely local the blood count would return to normal following removal of the teeth. Should the blood picture still remain abnormal, the question of some disturbance lowering the resistance would then appear to be present unless the pyorrhea had given rise to some secondary disturbance elsewhere. It was also desired to compare the changes, if any, following treatment by conservative means and by extraction to ascertain whether by conservative methods all infection can be eliminated.

Great care was exercised in the selection of patients; only those where the causative factor seemed obscure were selected. Examinations were made at the same time each day, thereby eliminating such factors as leucocytosis of digestion, exercise, etc.

The expected haematological results are well explained:

> It must be borne in mind that the reaction to infection is determined by the resistance of the individual. According to Welsh, the degree of leucocytosis is determined firstly by the nature of the toxin, and, secondly, by the nature of the leucoblastic reaction. We should, therefore, expect to find the most marked degree of leucocytosis where the infection is severe, along with a high resistance on the part of the individual. Patients with a low resistance can put up less defense, and hence we would expect a much less reaction. Also, in severe cases where the patient is unable to elicit any reaction at all, we may expect a little change in the blood; indeed, a leucopenia may be present. In such cases the toxin has paralyzed the reaction of the individual, rendering incapable any response.

A complete blood examination of each group is recorded. The cases were grouped, according to the degree of pyorrhea, as follows:

Group I—In its early stages, where gingivitis was present and pocket formation beginning.

Group II—Where pocket formation was established, but within the range of conservative treatment.

Group III—Advanced cases where extraction was indicated.

It would appear however, that nothing of definite character can be deduced from the foregoing. To attain results of any great significance, counts would have to be made of a great number of individuals, but we may draw from it the conclusions that in pyorrhea:

(1) There appears to be a decrease in erythrocytes and haemoglobin. The erythrocytes also show slight changes in size and shape.

(2) The polymorphs are decreased, while the lymphocytes are increased—a relative lymphocytosis.

(3) The mast cells show slight increase; such as is usually observed in mild, subacute inflammation.

(4) In the latter stages of pyorrhea the reaction is not so marked, which suggests that the prolonged absorption of toxins has weakened the resistance of the individual.

(5) Following treatment, the blood counts in a number of the individuals have returned to normal, which suggests to us that the lesion has been local. In the remainder the "relative lymphocytosis" has persisted. This may be due to some primary focus which has acted as a predisposing cause, or a secondary focus may have been established.

(6) The blood counts are typical of infection of a chronic nature.

—*J. A. Sinclair.*

285—Rachet, Marcel: **Buccodental manifestations of infantile acrodynia.** Thése de Paris, 878. 1937.

The beginning may be a gingivitis which has passed unperceived; or, at times, serious exfoliativé lesions. The author has observed twenty cases and makes special mention of the "exfoliative processes" of the alveolar tissue and bone, the teeth and their follicles. A very important chapter pertains to diagnosis, in which great emphasis is laid upon stomatitis as well as on simple gingivitis.

—*H. A. R.*

286—Schilling, Viktor: **Spricht eine Paradentose mit bestehender Leukozytose für bakterielle Genese und wie ist in diesem Falle die Therapie?** Does a paradentosis with an existing leucocytosis imply a bacterial origin, and in this case what therapy is indicated? Med. Welt (18):625. 1937.

Paradentoses are not associated with anomalies of the dentition alone. That the

general organism has a share can always be demonstrated. Paradentosis is often the result or accompaniment of an extraordinary process, whose notably infectious reaction is traceable to a focal endocrine disturbance or allergic circumstances.

A paradentosis may be accompanied by a gastro-enteritis, which has resulted from conditions in the oral cavity or which may be regarded as a parallelism to the paradentosis. In local treatment, the fundamental cause must be cleared up. By long observation of the course of the disease, it becomes possible to recognize the necessity of extraction.

287—Trauner, Richard (Wien): **Ueber Alveolen störungen mit tiefen Taschen.** Alveolar disease with deep pockets. Z. Stomat., **35**(9): 624. 1937.

Illustrations of deep bone pockets which occur only unilaterally (mostly buccal) on the teeth.

Causes: Penetration of a periapical suppuration into the tooth cervix, resorptions into the area of a retained tooth, perforation, osteomyelitis, cysts, osteitis fibrosa, epulis, root apex resection; or, from the gingival margin, traumatic occlusions and abrasions.

The diagnosis of such deep pockets in firm gingivae is difficult and is brought to light by use of fine, but not pointed explorers. The roentgenogram does not show a widening of the peridental cleft.

Treatment: Excision of the pockets. . . . If the pockets are too deep, only extraction remains.

288—Weichert, Johanna: **Klinische Untersuchungen über die Beziehungen zwischen Diabetes und Parodontose.** Clinical studies of the relations between diabetes and paradentoses. D. Z. Wschr. **40**(10): 211. 1937.

Fifty diabetics were examined. In 68% of the cases true paradentosis was established. This result is strongly opposed to those of other writers. The author explains this reversed relation on the ground that any appearance of disease in the periodontium was looked upon as paradentosis. For instance, *Butz* does not regard diabetic gingivitis as paradentosis; the author thinks he may be right.

—*Transl. of abstr.*
—*H. A. R.*

289—White, Cleveland J., M.D.: **Allergic manifestations in the oral mucosa.** J. A. D. A. **24**: 1521. Sept., 1937.

Allergy has become an important part of internal medicine yet it is seldom that allergic manifestations in the oral mucosa are discussed. Their diagnosis is "made by their clinical periodicity, the recurrence of lesions on use of or contact with these agents and their disappearance on the elimination of the suspected articles. The subjective symptoms are burning, tingling, and actual pain." The most common causative foods are chocolate, tomato, orange, egg, potato, and milk. Patients may become sensitized to any food or condiment.

The best method for detecting causative foods is to prescribe an elimination diet by using a basic nonallergic diet restricted for one week to coffee, tea, carrots, lettuce, prunes, plums, apricots, lamb, veal, rye krisp, olive oil, plum jam, and peppermint candy. Then if no new lesions add one food each day. New lesions will appear in a few hours after the offending food is eaten. This method is preferable to the skin tests in these cases of oral mucosa manifestations, when considering ingestants. The ingestion of certain medicaments may produce mouth lesions.

Reference is made to the work of Lain on the electrogalvanic phenomena caused by dissimilar metallic restorations, J.A.D.A. 23: 1641. Sept. 1936.

—*G. R. S.*

290—Winter, Leo, D.D.S.: **Differential oral diagnosis in blood dyscrasias.** (Editorial). D. Items of Int. **59**(3): 271-273. March, 1937.

Dental science is making such rapid strides that it is violating the time-honored tradition and pride of the dental profession that dental services differ from medical services in that our services are not a matter of life and death. This may have been true years ago when the judgment of the Doctor of Dental Surgery may not have been considered sufficiently important to jeopardize the life of a patient. This, however, is hardly true today. The opinion of the Dental Surgeon is quite serious in a case of Vincent's infection. Attention is called to the seriousness of agranulocytosis, anemia, leukemia or any other manifestations of blood disease. Can the dental profession now afford to be satisfied with our splendid record of past achievements and overlook the newer opportunities for greater services to this group of dental patients in need of our help?

Lack of dental judgment in the cases of a group of patients with blood diseases, some of them with fatal outcome, occasioned this

impressive and timely editorial. Two cases with fatal results are reported.

These cases are not of uncommon occurrence in the dental office. Unless these patients, according to Dr. Winter, are studied more thoroughly and the conditions more often recognized, with the diagnostic aids at our disposal, there is likely to develop a tendency which will bring the dental profession into disrepute.

"It should be noted that in many instances permission for post-mortem examinations are refused with the result that the hospital record certifies, and which is later interpreted accordingly, that the patient died following the removal of a tooth or teeth.

"Of course, the dental profession has every justification to place full confidence in the cooperation of the medical profession in this respect. The possibility of error in the medical or dental diagnosis is always present in spite of all precautions. The dental profession should, however, maintain its claim and constantly call medicine's attention to the possible relationship that exists between the teeth and their investing structures and the whole body. We must not overlook the further fact that in many instances the mouth, *especially the oral mucosa*, gives us the first and earliest sign of the disease, it is in fact the earliest evident expression of a systemic disturbance of a serious character in many instances.

In suspicious cases with mouth lesions, dentists should make it a routine procedure to insist upon a careful case history and, when necessary, employ laboratory examinations to establish a differential diagnosis. This especially applies to cases where suspicious mouth lesions are associated with conditions of pyorrhea, Vincent's infection, hypertrophy of the gums and pericoronitis. When the case history reveals the story of a pruritus, these precautions are bound to help the dentist and the patient by preventing the fatal dental outcomes in connection with blood dyscrasias.

—*J. A. S.*

291—Wissotsky ——— (de Tel-Aviv): Congrés dent. nationale. July, 1937.

W. presented:

1—Numerous observations and statistics demonstrating that periodontoclasia always accompanies endocrine disturbances. This is due to the fact that the tooth itself is an organ of internal secretion, most intimately linked with the general organs of internal secretion. Pathologic conditions produced in the pulp result in periodontoclasia. Hence, when the number of fusiform cells of the pulp is increased, we shall have paradentosis or pyorrhea. Numerous observations show that devitalization of the dental pulp in a pyorrhetic mouth notably decreases the intensity of the pyorrhetic process about these teeth.

2—There is a certain balance between the number of fusiform cells and the number of round cells found in the dental pulp. While this balance is maintained, the tooth and the periodontium will be perfectly intact. The rupture of this balance in one sense or another will cause:

a) increase of round cells—caries

b) increase of fusiform cells—periodontoclasia.

This seems to be the cause of the famous "antagonism" between caries and periodontoclasia.

—*H. A. R.*

(f) Treatment

(Continued from 8(2): 132, July, 1937)

460—Boyens, Paul J., D.D.S.: **Health reactions of San Quentin inmates to the treatment of paradentosis and gingivitis:** (Part of a symposium report on investigations carried out to determine the health reactions resulting from the surgical removal of gingival and paradental infections), in: J. A. D. A. 24(5): 685-90. May, 1937.

Surgical eradication was the treatment used in all cases. Number of patients operated on and rechecked, 70.

Only such systemic ailments were considered which might result from paradental infections and the health changes were noted after the infections were eliminated. No other treatment of any kind was given the patients therefore any improvement in general health conditions might reasonably be supposed to have resulted from the elimination of the infection. It is the common experience of periodontists to observe various forms of health improvement after the removal of paradental infection but medical and dental schools and practicing physicians and dentists have not given this fact the serious attention it deserves.

The cases were carefully selected and the survey covered a period of two years. The evidence submitted is largely clinical nevertheless this was supported by a consideration of certain laboratory findings. "Studies of the blood picture in paradental infections" is reported by Hilario G. Marquez, Ph. D., J. A. D. A. 24(5): 691-95. May, 1937. In Marquez' conclusions he states that, "Gibson's chart seems to indicate increased resistance of the host after treatment. Toxic degeneration of neutrophils as expressed by

fine granulation is decreased after operation; which indicates diminished toxic absorption. The incidence of indicanuria is less after operation."

"Paradental infections as related to prostatic infection": . . . J. A. D. A. 24(5): 696-98. 1937, was reported by George F. Oviedo, M. D. who states that "Although the number of cases is too small to be a basis for conclusions as to the role of paradental infection in prostatic disease, it is my firm belief that a definite relationship, although slight, does exist. When a ratio of ten out of nineteen infected cases, or more than fifty per cent, shows improvement, I believe that metastatic infection of the prostate does exist." "Paradental tissues are a frequent site of infection and a source from which pathologic bacteria are fed to other organs and tissues."

"Effect on the general health of treatment of paradental disease by surgery" by Charles F. Sweigert, M. D., I. C. Gobar, M. D., and Alexander G. Bartlett, M. D., J. A. D. A. 24(5): 698-702. May, 1937. After the operation nearly 100 percent of the men had more strength and better appetite and had gained weight. Many showed a definite improvement of headache and stomach disorders and reduction in fatigue, nervousness, and nocturia. After two years the conditions were about the same as one year after the operation.

The experimental survey is summarized by I. C. Gobar, M. D. in the same issue of J. A. D. A. as the above, pp. 705-6.

—*G. R. S.*

461—De Libouton: **Travaux de pratique courante** presented before l'Ecole Odontotechnique. (Paris): in: Rev. de Stomat. May, 1937.

In paradentosis (periodontoclasia), the mobility of the teeth is a constant factor and difficult to overcome. When it is desirable to relieve this condition a retention bridge should not be made too soon; but immobilization should be secured by means of wires. The author rejects the use of metallic wires but uses in his daily practice, braided threads of silk. These permit perfect local hygiene.

—*H. A. R.*

462—Dillon, Juan: **La chirurgie de la paradentose.** Surgery of paradentosis, in: Revist. Odont. **25**(2-4). Feb. to Apr., 1937.

The symptomatology of paradentosis (polyalveolysis) is discussed, especially the mucous and osseous lesions.

1—Gingivectomy. Methods of Ward, Crane-Kaplan, Howell, Sanders—all these technics permit alveolar curettage, removal of radicular tartar, polishing, etc., and suppression of suppuration. They have the special advantage of allowing the completion of the resection in two sittings.

2—Flap methods. Loosening of the gums, treatment of the root, replacement and suture of the flap. Only a small area (from 4 to 6 teeth) should be treated at a sitting; so that the treatment of the entire mouth requires about six sittings.

—*H. A. R.*

463—Gottlieb, Bernhard. (Vienna): **Die Ruhelage des Gebisses ausserhalb der Mahlzeiten.** The resting condition of the dentition outside of meals, in: Z. Stomat. **35**(5): 345. 1937.

In order to eliminate nocturnal grinding and to modify ("unload") the bite, it is recommended, especially between meals, not to bring the teeth together. At night, in sleep and on awakening, this should be under control. Holding the head far back is helpful in keeping the teeth apart.

—*H. A. R.*

464—Kohler, G.: **Problem of the fixation of the teeth in the treatment of paradentosis,** in: Svensk. T. Tidskrift. November, 1937.

The author rejects methods necessitating devitalization of the teeth. At the same time, he does not believe in the fixation of premolars or molars for which he advises extraction followed by restoration with partial prostheses in order to avoid danger of focal infection. He recommends, however, anterior retention of incisors and canines in almost every case.

Transl. of abs. in Rev. Stomat.

—*H. A. R.*

465—Nivard ———: **Les colloides dans le traitement des paradentoses.** Interesting observations in the treatment of inflammatory conditions with Selenium colloidale, in: La Rev. Odontolog. **59**(12): 1937.

In the treatment of buccal affections, the colloids develop a notable reaction, as in the treatment of other affections of microbic

origin, and in the treatment of septicemia.

Some hours after the first injection there is swelling of the gingival edge and the papillae; the proliferated epithelial granulation tissue of the pockets appeared to press outward from the pocket bottom like a rupture. More or less copious serous exudation was present. Sharp demarcation occurs between sound and diseased tissue, just as if the bone wished to throw off the diseased zone.

After 3-4 days, the reaction begins to fade and improvement takes place. The injections are perfectly painless; the patient has a subjective feeling of betterment from the first injection. The next injection 1 cc.: 3rd or 4th time 2 cc. are injected. On the whole at least 10 injections are made, with perhaps two or three sprayings each week.

Bacteriologic control shows a rapid lessening of numbers in cultures.

—*H. A. R.*

466—Nye, R. N.: **Relative in vitro activity of certain antiseptics in aqueous solution,** in: J. A. M. A. **108**(4): 280. Jan. 23, 1937.

The author experimented with certain proprietary and non-proprietary antiseptics in ordinary use. The tests were made *in vitro* and in the presence of blood lymph, tissue juices, and secretions of the mucous membranes, so as to approximate conditions in the body. Four iodine solutions of different strength and both aqueous alcoholic were tested; seven containing mercury (metaphen 1:500, merthiolate 1:1000, mercurochrome 1:50, among others), two containing chlorine; also hexylresorcinol 1:1000, listerine (25% alcohol) and pepsodent. The experiment seems to prove the superiority of iodine as an antiseptic; even in as weak an aqueous solution as 1:128 it was more effective than the other substances tested. Moreover it fulfills admirably the prerequisites of a satisfactory antiseptic, being readily diffusible, not unduly toxic and inexpensive. Nye thinks antipathy to iodine may be because of too strong solutions ordinarily used and because of irritating effect of alcohol content, which is eliminated of course by using an aqueous solution.

467—Rosenthal et Pierron: **Traitement des pyorrhées, gingivites, paradentoses, par des injections intraveineuses de fluorure de calcium en suspension magnésienne.** Treatment of pyorrhea, gingivitis, paradentosis, by intravenous injections of flouride of calcium in a suspension of magnesium, in: Rev. de Stomat. **6**: 435. 1937.

In all cases treated by these authors, there was obtained:—

1) Arrest of gingival hemorrhage. 2) A healthy appearance of the mucosa. 3) Arrest of gingival pain. 4) Tightening of the teeth.

Local treatment was only resorted to if the mucous membrane was not in alignment at the alveolar margins.

Three observations of bony consolidation, partial, very clear, in spite of the persistence of suppuration seem to prove that the authors have found in the fluoride of calcium in suspension of magnesium a most powerful recalcifiant.

—*H. A. R.*

468—Sorrin, Sidney, D.D.S.: **Bone changes following conservative periodontal treatment,** (Abstr. 14), in: J. D. Res. **16**(4): 302-3. Aug., 1937.

Cases varied in character as to age, sex, occupation. Pocket formation, suppuration, mobility present before therapy. Pocket formation determined by application of probe into pocket. After treatment, pocket closed, suppuration ceased, teeth much firmer, and gingival tissues acquired healthy color. Impossible to pass probe into areas where pockets previously existed. Interesting bone changes noted by roentgenographic examination. Patients under observation for many years following treatment; with few exceptions, no recurrence. Bone regeneration clearly demonstrated; periodontal tissues remained in good health since completion of treatment —usual establishment of occlusal equilibration, careful instrumentation of hard and soft tissues in pocket, and instruction in gingival massage and diet. No systemic complications. Pinledge splints, continuous-clasp dentures, and ligatures most helpful in effecting cure, because of assistance in immobilizing teeth.

—*Copy.*

469—Sorrin Sidney, D.D.S.: **X-ray changes following treatment of osteofibrosis,** (Abstr. 51), in: J. D. Res. **16**(4): 321. Aug., 1937.

Case of osteofibrosis showing improvement after local therapy—occlusal equilibra-

tion. All systemic examinations negative. Biopsy of tissue about lower right cuspid tooth diagnosed as osteofibrosis. Teeth responded to normal vitality tests. Occlusion balanced and denture constructed. One year later, osteofibrosis areas improved, but new areas appeared. After further adjustment of denture, new areas disappeared. Denture utilized more teeth by securing greater reciprocal anchorage. Systemic examinations negative. Author indebted to Drs. Darlington, Schuyler, Swenson, Winter, Miller, Greenfield, Salman, Wald and Trapozzano for help and coöperation.

—*Copy*

470—Szabo, J.: **Das Paradentose problem (Pyorrhoe) vom allgemeinmedizinischen Standpunkt.** The problem of paradentosis (pyorrhea) from the standpoint of general medicine, in: Münch. med. Wschr. **84**(1): 35. 1937.

Since cure is impossible, according to the author's pessimistic view, prophylaxis is the next most important subject. For this, thorough research is indicated at the onset of the earliest symptoms, as well as skilled treatment at the hands of the dentist.

—*H. A. R.*

471—Thibault, R.: **La gingivectomie: Gingivectomy,** in: Rev. de Stomat. **39**(11): 1937.

Gingivectomy is indicated:

1) When it is desired to eliminate the pyorrheal pockets quickly; for instance in the course of general accidents with buccal septicity, or before surgical intervention related to the digestive tract, to prevent the swallowing of pus.

2) When other methods of treatment—caustics, vaccines, sclerosing injections, etc.—have proved ineffective.

The technique of after treatment is discussed and toothbrushing and massage recommended.

—*H. A. R.*

472—Vantier, U.: **Contribution to the therapy of the paradentoses.** Thése, in: Rev. mens. suisse d'Odontologie, **11**: 1097. Nov., 1937.

Article is devoted chiefly to a résumé of methods of prominent specialists; Roy, Hulin, Gottlieb, etc. The author finds the method of Neumann the best, i. e., radical surgical intervention: after a critical review of different treatments for pyorrhea and reports of 39 observations.

V. thus defines what is meant by cure:

Clinically—gum must be healthy, free from pockets, clasping the root with a firm fibrous ring.

Radiographically—bone must show a free border of regular contour with tendency to recalcification and to the disappearance of enlarged periodontal spaces.

Cure persisting after one year may be considered final, provided that the patient preserves a rigorous hygiene and good general health; hence the need for medical collaboration. From the statistics the author concludes that each method finds usefulness according to the degree of the disease and the character of the patient. He adds: "to know this, it is up to the practitioner to improve his technique and his chances of success. However, as we have shown, radical intervention offers the greatest number of chances of cure in the cases of inflammatory paradentosis. It goes to the root of the lesions, permitting curettage of all the inflamed tissues of the paradentium, inciting cicatrization and permanent consolidation, whatever the constitutional etiology."

—*Transl. H. A. R.*

(g) Home Care and Prophylaxis

(Continued from 7(2):122. July, 1936.)

63—Albert, Leo, D.D.S.: **Advancement of the toothbrush,** in: D. Cosmos, **78**: 459-467. May, 1933.

The author describes several of the older methods employed in brushing the teeth. These include the vertical and the rotary. In view of the fact that these have proven detrimental to the surrounding gingiva and often useless from a prophylactic standpoint, he provides his own method. He suggests.

(a) labio-lingual brushing—brush at 45° angle, small vibratory circular motion, brush held parallel to the plane of the teeth.

(b) occlusal brushing—same circular motion, bristles perpendicular to the surface; sweeping by means of spiral motion food is kept out of tooth crevices.

The benefits derived from this method are:

1) toning of the gingiva
2) ease of movement makes for speedy action
3) obviation of hemorrhage and laceration

This method takes the place of the need for hard food for natural gum massage. It is a prophylactic measure for dental tissue preservation.

Child education, literature distribution, lectures, etc., should all be used to draw the public eye to the need of a toothbrush and the proper method of toothbrushing.

Education by the practitioner at the chair is very important. Trouble that could have been avoided, correction of bad brushing habits, and instruction in the proper method, a simple and orderly one, will gain permanent adherents.

An ideal toothbrush is described:

1) straight handle
2) five to six rows of bristles
3) three rows abreast
4) height—½ inch; width—5/6 inch
5) all uniform in size
6) bristles should not be soft or yielding
7) same formula, small size, for children

He gives the following order of brushing:

1) upper jaw first
2) divide arch into quarters—facial surfaces first; lingual and occlusal completing the section
3) overlap contacts of brush areas to avoid omission of any particular section
4) change of position necessary for the anterior lingual, tipping as required towards the tip of the brush.

Limitations of the method are:

1) failure to cleanse completely in the interdental spaces
2) gingival trough is not reached
3) antero-lingual still difficult to cleanse
4) haste and negligence reduce the efficiency of this method as well as of any other.

64—Badanes, B.: **The dental finger cot versus the toothbrush in gingival massage,** in: D. Cosmos, **78**:173-177. 1936.

Dentally speaking, our civilized life compares unfavorably with that of our ancestors. The refined foods of today prevent us from receiving the benefits to be derived from chewing on solid food and the resulting stimulation of the paradentium from such action. Deficency diets which will experimentally produce caries will when fed to the same animal in the form of hard biscuits show no demonstrable tooth decay or destruction of surrounding tissues. Our ancestors used no toothbrushes but their diet of solid natural foods maintained a healthy oral state and automatically cleansed their teeth. As a result dental disorders were less common in those days. Dietary experiments pay too much attention to the composition of diets and not enough to the function of solid foods in tissue stimulation.

Inasmuch as a clean tooth will decay it is obvious that the function of the toothbrush is not mere scrubbing of the teeth but rather tissue stimulation. To use the brush properly requires undue skill which the general public does not seem to possess. Furthermore, in the hands of the inexperienced the toothbrush can do more harm than good.

The author believes that the finger cot offers the public a simpler and more effective means of soft tissue massage than does the toothbrush. Such massage increases the nutritional blood supply to gums and pulp and liberates tissue stimulating hormones thus increasing the functional condition of the tissues and the vitality of the tooth pulp.

—*G. H.*

65—Bricker, Frederick A., D.D.S.: **Importance of mouth brushing,** in: J. A. D. A., **23**:2287-90. 1936.

Educating the patient in the importance of his part in mouth care is presented as essential to a dentist's achieving results. While mouth brushing is not all-important in the prevention of disease, an unclean mouth plays a great part in encouraging it, and patients should be shown how to keep their mouths as clean as possible. Interest can be aroused most readily by presenting actual models, mirrors, and toothbrushes. Allow the patient to see roentgenograms of his own case and follow these by an educational program in several parts. The following points should be discussed in full:

(1) The local causes of decay and periodontal disease.

(2) The local causes of periodontoclasia.

(3) Prevention of decay and periodontoclasia.
(4) Coöperation of the patient.
(5) The mouth as a seat of infection.
(6) Fundamentals of diet as affecting teeth.

Emphasis is placed on a discussion of the general circulatory system and its relation to the circulation of the blood in the mouth. The patient is told how, by brushing, he may aid and increase this in his own mouth.

In reply to the most common questions asked by the patients: A stiff brush is advocated and the patient is advised to brush as hard as he can without causing discomfort.

The first symptoms of periodontoclasia that a patient will notice are inflammation and bleeding of the gums and sensitiveness to the brush; looseness of the teeth and tenderness in biting in the more advanced stages.

A powder is recommended in preference to a paste. An individual is prone not to rinse his brush when using a paste in an attempt to make one brushful do its work over the entire mouth. The brush becomes filled with plaques and the remaining paste is forced down into the bristles where it is entirely useless. It is a very simple matter to dip the tips of the bristles into the powder for each section of the mouth the length of the brush itself. The result is a more thorough cleansing. Systematic and thorough brushing is stressed as being more important than mere frequency which, by itself, brings no results.

—*B. G. S.*

66—Grove, O. H.: **Gingivitides and oral spirochetoses.** Med. Bull. of Veterans Administration, Washington, D. C. Oct., 1937, p. 164. Abstract in J. A. M. A., **109**(19):1580. Nov. 6, 1937.

Oral hygiene is most effective method of diminishing incidence of gingivitis and Vincent's angina. If tissues are broken or continuity of epithelial cells disrupted in any way surgical asepis is important. In pyorrhea cases 85% of curative work depends on patient's oral hygiene habits. It would seem necessary that patients use penetrating alkaline mouth wash daily to aid in removal of debris harboring micro-organisms. Same organisms are responsible for gingivitis and pyorrhea, Vincent's infection, noma and Ludwig's angina. Mercury (in form of calomel), as systemic spirocheticide, proper elimination by use of salts, and H_2O_2 as local oxidizing agent offer simple, agreeable and effective treatment for Vincent's spirochetoses. Often people under regular dental care have been allowed to develop pyorrhea to a stage where systemic involvement makes saving teeth impossible.

(h) Vincent's Infection

(Continued from 7(2):126. July, 1936.)

220—Christie, David, M.D. (Saskatchewan): **Vincent's stomatitis and granulocytopenia,** in: J. A. M. A. **108**(19):1669. May 8, 1937.

In letter to editor Christie expresses surprise that Farrell and McNichols make no mention of granulocytopenia in their article on medicaments in treatment of Vincent's (in J. A. M. A. Feb. 24, 1937). Leucocyte and differential blood count is always necessary in severe stomatitis regardless of kind of organisms present. Farrell and McNichols report three cases of noma without mentioning blood count. Certainly before using neo-arsphenamine blood should be examined. Investigation should not be considered complete with discovery of Vincent's organisms, as granulocytopenia may be present also.

221—Clark, D. R.: **Vincent's infection,** in: J. A. D. A., **23**:1345. July, 1936.

The author presents a valuable and complete review of Vincent's infection. The etiology, contemporary theories, pathologic picture, both gross and microscopic, symptoms, clinical picture, and methods of treatment are all given adequate consideration.

222—Farrell, G. W. and W. A. McNichols: **Efficacy of various medicaments in the treatment of Vincent's stomatitis.** J. A. D. A., **24**:835. May, 1937.

The authors report treatment of 794 cases of Vincent's stomatitis; some with serious complications and six ending fatally. After a study lasting two and one-half years, in an institution where Vincent's stomatitis is common, the authors conclude that the severity of a case is determined by the resistance of the individual and the virulence of the infecting organisms. Irritation from poor-fitting dentures, malocclusion, broken-down teeth and smoking seem to lower resistance to infection. In all cases, no mat-

ter what treatment is employed, a thorough prophylaxis is essential if the cure is to be lasting. Abstinence from cigarette smoking and alcohol is recommended during the infection. No surgical procedures are to be attempted. The authors report they were called to see three patients who were dying, following extraction of teeth in the presence of a mild Vincent's infection. In most cases the disease becomes more acute the longer it is allowed to progress without treatment. Solution of hydrogen peroxide, U. S. P., is the prime factor in curing Vincent's infection. The authors used it full strength four times a day.

Neoarsphenamine and glycerine, 10 per cent, was used in 65 cases. In half of this number the condition cleared up promptly. The remaining portion of this group were then treated with the arsenic plus hydrogen peroxide, and all were cured.

The authors tried 15 per cent chromic acid in 281 cases, without success. Other drugs used and discarded were aconite, iodine and chloroform (140 cases) aniline dyes (110 cases) and compounded tincture of benzoin (35cases.)—*J. A. M. A.*, 108:631, February 20, 1937. —*M. K. Hine.*

223—Lyons, D. C.: **Public health significance of Vincent's infection,** in: D. Digest, **42**:150. 1936.

The author describes Vincent's infection and mentions the organisms responsible for it, borellia Vincenti and a spirochete.

In the chronic form of Vincent's the spirochetes predominate, whereas in the acute form the two are almost of equal number. The agent producing the sloughing and pain is the bacillus; the agent penetrating the tissues is the spirochete.

Chronic Vincent's is a public health problem for individuals infected with it are carriers. Organisms comparatively harmless to the carrier are often virulent to those infected. Vincent's should become one of those diseases requiring a report to the authorities upon discovery and subject to certain limitations and sanitary measures. Late reports indicate that this disease is increasing in prevalence. The major cause of increased incidence has been traced by the author to beverage-dispensing establishments. Better control of Vincent's and the education of the public to demand proper sanitary measures are warranted public demands.

224—Maxwell, C. H.: **Treatment of Vincent's infection with acetarsone,** in: N. Y. State J. of Med., **36**:843-898. 1936. Abstr., in: D. Survey, **12**:424. 1936.

The continued prevalence of Vincent's infection, the intractibility of the disease and the difficulty of successfully treating it, has prompted the use of countless drugs and compounds in its therapy.

The latest addition to the therapeutic armamentarium of the clinician is acetarsone, an arsenic compound, used successfully by Maxwell to treat 74 cases of Vincent's infection. Twenty-eight patients received the drug by mouth; 36 were treated with it by mouth plus the use of a mercurial wash; 10 were treated only with acetarsone by ingestion and as a paste massaged into the affected parts. All improved rapidly. One other case improved but was not cured by the drug.

225—Nesson, J. H.: **Diagnosis and treatment of Vincent's infection,** in: J. A. D. A. **23**:1483-1492. 1936.

After giving some historical and bacteriological background, the author proceeds with etiology. Leading investigators believe that steptococci initiate the lesions and pave the way for the saprophytic, gangrenous action of the fusospirochetes. Fusospirochetes are normally present in the mouth. Lowered resistance gives them their hold. The infection is primarily found in unclean, pyorrhetic mouths.

Regions where favorable incubation is found are:

1) Pericoronal flaps
2) Buccal gingival margins of the second and third molars in close contact with the cheek mucosa.
3) Deep periodontal pockets; palatal crevices of approximal gingivae of upper central incisors
4) Areas of faulty restoration (crowns, fillings, etc.)
5) Tonsils
6) Improper contacts
7) Areas of irritation-smoking, calculus, traumagenic occlusion

Treatment of Vincent's belongs entirely to the dentist.

Nutritional factors in inciting Vincent's infection are:

1) Avitamininosis

2) Malnutrition (lack of calcium and phosphorous)
3) Disturbed metabolism

Systemic factors in inciting Vincent's infection are:

Scurvy, cancer, syphilis, alcoholism, diabetes, fatigue, dissipation, colds, and blood dyscrasias.

Communicable factors are:

Kissing, towels, drinking cups and glasses.

Between the years 18-30 individuals are most susceptible.

The author lists all the clinically observable symptoms.

Treatment by means of chromic acid, if properly applied, is one of the most effective means for eradicating this condition. This drug is universally used. The detailed method for using chromic acid follows. When the acute symptoms have been alleviated bland dyes such as 1% acriviolet or tincture of metaphen are applied.

Having relieved the symptoms, the predisposing causes to this condition should be studied.

Editor's note:

(The reader is referred to "Effects of medicaments on the motility of the oral flora" by S. Leonard Rosenthal, J. of Periodontology 8 (2): 71-80, 1937.)

226—Rose, E. D.: **Vincent's infection, diagnosis, treatment, and prevention,** in: J. Am. Hyg. Ass'n., 10(2):15-19. 1936.

The etiology and clinical symptoms of Vincent's disease are briefly reviewed. The contagious and infectious nature of the disease is stressed. Mere destruction of the bacteria involved leaves periodontal pockets which must be effectively removed.

Instead of employing oxidizing agents in the treatment of the acute phase of the disease, the author recommends the use of sodium tartrate buffered with sucrose. The drug is administered intramuscularly into the biceps, gluteal or deltoid muscles. Three doses are given daily:—2c.c. dose of a 1.5% solution. The acute condition will clear up within ten days. Mild laxatives, etc., are used to build up general resistance. The acute stage cleared up, curettage and prophylaxis are carried out. Dietary regulation, including sufficient orange juice, is an important factor in treatment.

—*G. H.*

227—Tishler, B.: **The general practitioner's misconception of Vincent's infection,** in: D. Cosmos, 78:178-180. 1936.

Dr. Tishler is of the opinion that many practitioners do not understand the clinical and bacteriological syndromes of Vincent's disease. Many dentists, in cases of gingival hemorrhages send a blood smear to a laboratory and upon the report of a positive Vincent's make a diagnosis of Vincent's. Many such cases exhibit none of the classical signs of acute Vincent's, i. e., ulceration, pseudomembrane formation, interpapillary destruction, etc., and are termed chronic or subacute Vincent's. There is no such thing as chronic or subacute Vincent's. The disease is an acute one.

The mere presence of the fusospirochetal symbiosis is not sufficent evidence for a Vincent's diagnosis. Vincent's smears may be obtained from gingival hypertrophy and periodontoclasia. The so-called subacute type of Vincent's is usually due to chronic periodontal disease.

Spontaneous hemorrhage is symptomatic of periodontoclasia and should not always lead to a Vincent's diagnosis. Treatment of such hemorrhage symptomatically is futile if the underlying causes are not removed. The author emphasizes the presence of ulceration as an adequate sign of Vincent's and the fact that chronic periodontal disease involving the entire mouth is often mistaken for Vincent's.

—*G. H.*

(i) Miscellaneous

(Continued from 7(2):134. July, 1936)

167—Adloff, P.: **Das biologische Gleichgewicht im Lueckengebiss.** The biologic equilibrium in a dentition where teeth are already extracted. Z. R. 16:657. 1935.

The articulation equilibrium is dependent upon many conditions. Adloff believes that it is not disturbed as much by spaces caused by loss of teeth as always believed. Nature always tries to restore a state of equilibrium. In many cases sufficient masticatory work is possible even though many teeth are missing.

168—Balendra, W.: **Betel chewing in relation to oral sepsis and carcinoma,** in Antiseptic, 32:570, 1935; abstr. Am. J. Cancer, 26:426, 1935. J. A. D. A. April, 1937.

About 90 per cent of the whole population of India and Ceylon indulge in betel nut chewing and 94 per cent of the patients with oral carcinoma are addicts. Of the malignant conditions observed in the mouth, about 90 per cent were on the mandible and adjoining teeth, 8 per cent on the tongue and 1 per cent on the lip. The teeth of addicts become brittle and sharp through erosion and irritate the soft tissue. As the betel is often chewed with tobacco leaves, another factor of irritation is added. The process leading to malignancy is characterized by extreme slowness, few persons developing true malignant growths in less than from fifteen to twenty years.

—*Hamilton Robinson.*

169—Blankenhorn, M. A., and Richards, C. E.: Garlic breath odor. J. A. M. A. 107(6):409. Aug. 8, 1936.

Lehmann in 1930 first reported a study of alliacious oil odors in the body, concluding that odors pass into the blood stream, are aerated from the blood to the lungs and thence into the expired air, but recently Haggard and Greenberg, (J. A. M. A. June 15, 1935,) have made tests and arrived at the conclusion that "this odor arises solely from particles of onion and garlic retained in the structures about the mouth." Blankenhorn and Richards' recent experiments refute these findings. They administered foods containing garlic to four patients who because of surgical condition ingested food and breathed through separate openings, and in all of these the odor of garlic could be detected on the breath. This odor was not overcome by washing the mouth with 1% chloramine, as has been suggested, except for a comparatively short time and then apparently was only disguised. The authors admit that purely scientific chemical tests of the intensity of the odor were impossible but are convinced that "garlic and onion breath come from the blood by way of the lungs."

170—Bliss, S.: Causes of sore mouth in nephritis. Abs. (Abbreviated), in: J. of Bio-Chemistry, Nov., 1937, in: J. A. M.A. 110(3): 240. Jan. 11, 1938.

Severe ulcer of cheeks and tongue in nephritic patients and nephrectomized dogs may be a local ammonia irritation. Bliss found dogs' tartar to contain urease. Ammonia is formed when urease acts on salivary urea. Dogs with normal blood non-protein nitrogen have no ulcerations even from tartar, but just where tissues touch teeth in nephrectomized dogs there is ulceration. These ulcers clear on removal of the tartar though blood non-protein nitrogen remains elevated. Tartar from normal nephrectomized dogs hydrolyzes urea solution to ammonia in a few minutes (as does urease). He demonstrated that ammonia actually damages soft tissue.

171—Breuer, Karl: Ueber die Erkrankung des Paradentiums des Splenomegalic Typus Gaucher. Paradental disease with splenomegaly of the type Gaucher, in: Z. R. 24:1064. 1935.

Breuer reports four cases of Morbus Gaucher (a disease of the lymphatic and hemopoietic organs with enlargement of the spleen) where marked changes in the paradentium were present. After removal of the spleen, the symptoms in the paradentium disappeared. They are of secondary nature and in most cases inflammatory.

172—Council on Pharmacy and Chemistry of the A. M. A. Shotgun vitamin therapy; in: J. A. D. A. 23:491-494. March, 1936.

The public has become "vitamin-conscious" and to satisfy their requirements for vitamins, manufacturers have put many preparations on the markets, even though there is no need for them when one follows a well-balanced diet. Attempts have been made to artifically combine two or more vitamins in one prescription, with the result that one or all of them lose their original effectiveness. A few experiments have been made to prove this and in no instance have the expected results been achieved. There is, however, very little information in this field and the manufacturers themselves are the only ones who can prove such claims as are made about their products. It has been questioned as to whether or not there might be a great value if one vitamin is combined with another but nothing has been written to support this theory, except as the vitamins appear together in nature. Most diseases only require an added quantity of one vitamin without the increase in the intake of another and in giving a combined dosage the excess of the latter might prove harmful.

In preparing vitamins for artificial dis-

tribution, often much of their potency may be lost through oxidation, combination with mineral oil as well as with minerals, heat and other chemical reactions. Some attempts have recently been made to stay this destructive activity, but it is thought that the effectiveness may still be lost through standing in combination, thereby rendering both ineffective or, at any rate, requiring a larger dosage.

The conclusion reached from the above evidence is that there is a great need for further research on the question; that so far there are no definite conclusions to be drawn.

173—Dunnington, J. H. and Khorazo, D.: **Conjunctivitis due to fusospirochetal infection,** in: Ann. of Den., 4:214. Dec., 1936.

A case of conjunctivitis is described in which the condition apparently was due to a fusospirochetal infection. There was no evidence of such infection in any other part of the body. Protracted treatment with copper sulphate and 1% solution of silver nitrate brought no improvement. When application of 1% solution of sodium perborate was added the condition rapidly improved.

174—Editorial. **Potassium chlorate in dentifrices,** in: J. A. M. A., 108(16): 1343. Apr. 17, 1937.

Editor comments on article by Richardson in *Journal Pharmacol & Exper. Therap.*, (60: 101, 1937) in which he assails the use of potassium chlorate in dentifrices stating that their prolonged use might result in absorbtion of enough chlorate to injure the blood, kidneys and liver. Such harmful effects he found due to the chlorate ion not the potassium ion. One dentifrice in general use contains 41% potassium chlorate and deaths have been attributed to the accidental swallowing of large amounts of this preparation. While it is not necessary to swallow dentifrices and not intended that one should do so it is obviously irrational to use a substance with such possibilities particularly since the salt effect may be got advantageously from harmless sodium chloride.

175—Haggard, Howard W., M.D.: **Eliminating odor of garlic.** in: J. A. M. A. 107(11): 895. Sept. 12, 1936.

Discussing the report of Blankenhorn and Richards (J. A. M. A. Aug. 8, 1936) Haggard says that they merely repeated Lehmann's experiment on which he and Greenberg have previously commented (J. A. M. A. June 16, '35.) Haggard and Greenberg gave garlic in amounts "within reason" and no appreciable amount appeared in the blood stream, while Blankenhorn and Richards, he says, administered "large quantities." When Haggard administered garlic in sealed gelatin capsules there was no breath odor. The simple experiment of chewing the garlic and spitting it out without swallowing would prove that the oil need not get into the blood stream before being detected on the breath. Haggard and Greenberg have made further tests with deodorants and find a dilute solution of sodium hypochlorite somewhat more effective against the odor than chloramine, previously recommended. The hypochlorites destroy garlic odor in test tube as well as in the mouth, and have themselves no persistent odor that would disguise or "mask" odor of garlic (as claimed by Blankenhorn and Richards). Only the substances which chemically break down odoriferous substances are effective deodorants. Hypochlorites have no effect on the breath odor from alcohol which definitely eminates from the blood stream.

176—Healy, J. C., and Dailey, F. H., and Sweet, Marion H.: **Medical aspects of periodontoclasia and gingivitis,** (Abstr. condensed). in: J. A. M. A. 106(24): 2105. June 13, 1936.

In a study of 86 cases of various types of periodontoclasia and 26 cases of gingivitis of various types, investigation of the history revealed the probability of some form of allergic disease in 46 of the former and 16 of the latter. In allergic periodontoclasia, the capillary dilation, increase of tissue fluid, and spasm of the arteries and arterioles account for the primary alteration of the gingivae and the formation of calculus. With continuation of the arterial spasm, the vessels become sclerosed, the blood supply is insufficient, and there is atrophy and breaking down of the peridental fibers, which cannot be replaced because of lack of nutrition. Local treatment gives temporary relief, but permanent improvement is rare. There were 10 cases of gingivitis of pregnancy studied and 6 presented local gingival eosinophilia and all these gave positive skin reactions to proteins and were allergic to

lactobumin, lactoglobulin, or casein. Since milk proteins are very common allergins, the theoretical necessity of large amounts in the gravid woman's diet may be questioned. In summarizing these cases of allergic nature it may be said that they have certain diagnostic features in common: eosinophilia, capillary dilation, increased permeability, and exudation. Other important observations: history of food idiosyncrasies, familial predisposition, positive skin reactions and skin tests with urinary proteose.

177—Koch, F.: **Karies und Paradentose** in Brasilien. Parad. 8: 161. 1936.

Brazil offers a great field for the observation and study of the differences in the occurrence of paradentosis and caries among the different races of the rural and urban population. Dental conditions are the better among the rural population the purer the race is. The negroes have the best teeth, while the races from north and central Europe have the worst. Among the urban population the lower class which is usually racially very mixed, has the worst teeth, because dental care is practically unknown to them. Following means are recommended as prophylactic measures: systematic ultraviolet radiation of weak and young dentitions, systematic nourishment with hard food in early childhood. and the introduction of salvadora persica branches such as used by African negroes for their dental care.

178—McCarthy F. P.: **Etiology, pathology and treatment of leukoplakia.** Report of 316 cases in: Arch. Derm & Syph., 34:612. Oct., 1937. From abstract J. A. M. A. 107: 1838. Nov. 28, 1938.

Author's investigations indicate that in extensive leukoplakia of the tongue the predisposing factor is usually untreated or inadequately treated syphilis. Elsewhere in the mouth there is apparently no connection between syphilis and leukoplakia. Constitutional factors, lack of vitamin A or a hereditary predisposition to atrophy or other tissue degeneration may be etiologic factors. Except for the preventive form of treatment (removal of irritating conditions) the treatment of leukoplakia is unsatisfactory. The use of radium and caustics is contraindicated, as there is a possibility of initiating malignant change in the lesion. Surgical removal of the malignant lesion is indicated in some cases, but unless the local irritation is eliminated, leukoplakia will recur.

179—Ormerod, F. C.: **Tuberculous ulceration of mouth and pharynx,** in: J. of Larynogology and Otology. Sept., 1937. Abstr. (abbreviated) from J. A. M. A. 109(22): 1856. Nov. 27, 1937.

Tuberculous disease of mouth and pharynx is usually a complication of severe disease in lungs and is a bad sign found mostly in young patients from 20 to 30 years of age. Ulcers are flat and not deep with an irregular slightly undermined edge; base is granular and covered with tenacious mucous, which is important in diagnosis.

180—Prime, James Mark, D. D., Sc.: **Is operative dentistry failing?** in: J. A. D. A., 47-58. Jan., 1936.

The author states that the great percentage of dentists is interested in the replacement of the teeth rather than in the saving of them. Millions of teeth are sacrificed because operative dentistry has failed in its purpose. Deficient diet may play a part in tooth decay, as well as the failure to use the toothbrush properly. However, he believes that operative procedures are the most effective means to insure results.

Prophylactic treatments must be repeated often; a healthy periodontium is not retained without care. The operative dentist and the periodontist must work hand in hand. The periodontist cannot save a tooth which is being destroyed by caries; neither can the operative dentist save one if the periodontium is being destroyed by periodontoclasia. "Failure to recognize and properly treat periodontal lesions in their early stages constitutes an indictment equal, in some respects, to that of operative dentistry."

—*G. R. S.*

181—Query: **Excessive dryness of mouth,** in: J. A. M. A. 108(12): 997. Mar. 20, 1937.

Patient with inoperable cancer of breast and metastases to liver suffers from excessive dryness of mouth and tongue in spite of taking sufficient liquid and in absence of general dehydration. Various chewing gums, lozenges, etc., have been tried without relief, and editor recommends trying 1% pilocarpine. Care must be used to determine indi-

vidual sensitivity to the pilocarpine before settling on frequency and amount given.

182—Query: **Excessive salivation.** J. A.M. A. **109**(4): 299. July 24, 1937.

For excessive salivation in connection with wearing of full denture it is suggested as a possible remedy that carbohydrates be reduced in the diet and fruit juices and liquid in-take be increased. Aside from this there seems little known about the condition.

183—Query: **Sensitivity to podophyllum.** J. A. M. A. **109**(2): 155. July 10, 1937.

A case of severe ulcerative gingivitis followed administration of capsules of unknown composition. Blood pictures, smears and cultures were negative and podophyllum was suspected because it had been found in a medicine prescribed in similar case; editor, however, thinks this diagnosis unlikely, having found no record of anything of the kind after exhaustive research although one case was recorded of keratitis and dermatitis produced by dust of the root of podophyllum. These resinous cathartics are practically unknown as causes of either dermatitis, stomatitis or gingivitis. The cause was probably some other medicament in the capsules. A list of drugs likely to cause lesions of the mucous membrane is to be found in Prinz and Greenbaum's *Diseases of the Mouth and Their Treatment* (Lea & Febinger, Philadelphia 1935) and in Zinsser's chapter of Josep Jadassohn's *"Handbuch der Haut und Geschlechtskrankheiten"* Vol. 14 No. 1.

184—Query—**Painful tongue.** J. A. M. A. **106**(26): 2256. June 27, 1936.

Advice is asked concerning an elderly female with a smooth, very painful red tongue without visible ulcerations. Such a tongue associated with a hyperchromic anemia and a 1.5 color index as in this case, is frequently seen in pernicious anemia. Complete blood studies and blood chemistry studies, including non-protein nitrogen, sugar, creatinine, and serum proteins should be made. If the proteins are low, adequate intake should be provided; also liver in some form or dilute hydrochloric acid after meals, or both, may be of value.

185—Query: **Pseudocolloid of the lips.** J. A. M. A. **107**(16): 1326. October 17, 1936.

Lesions in pseudocolloid of the lips are likely due to presence of aberrant sebaceous glands in the buccal mucosa. They may be destroyed with cautery point or with the cautious use of X-ray, or radium, or carbon dioxide snow. The condition is harmless.

186—Query: **Burning of the tongue,** in: J. A. M. A. **106**(5): 403. Feb. 1, 1936.

Pregnant patient suffers from burning tongue enlarged to the extent that the teeth leave impressions in it. Tongue is deeply fissured and blebs form on borders and tip. Tongue was considered unusually large at birth and painful condition has recurred at various periods. Teeth are in good condition; tonsils in good condition except for inflammation of anterior pillars. Patient is of nervous temperament.

Answer: Pregnancy may have caused temporary increase in lymphangiomatous tissue which may have been present since birth, or it may be of neurotic origin, control of which condition is little understood. Burning is probably due to pressure. Two per cent $AgNo_3$ applied to fissures and blebs once a day, and Dobell's solution, diluted with glycerine as a mouth wash, are suggested. If condition fails to respond and swelling interferes with speech and swallowing, multiple fine punctures or excision of wedge from tip, followed by suturing, might be considered. Improvement might follow tonsillectomy.

187—Greenbaum, Sigmund, M.D., Philodelphia, Pa.:**Burning tongue:** Correspondence in : J. A. M. A. **106**(11): 940. March 14, 1936. Referring to reply to foregoing Query (J. A. M. A.) (Feb. 1, 1936).

Greenbaum states that "Burning Tongue" is observed in various states, as simple glossodynia exfoliativa, the avitaminoses, the anemias, the irritated and inflammed grooves of a scrotal tongue and Lain's disease. The grooved tongue described is not unusual with congenital macroglossia and the dental imprints sometimes produce a "lobulated-tongue." Food collects in the deep furrows, decomposes, and the irritation, and perhaps infection, cause burning. If thoroughly and frequently cleansed with sodium perborate, healing is usually prompt. There is also "burning tongue" in lingual tonsillitis, Costen's temporo-mandibular derangement, in cancerophobia, and lingual papillitis. Greenbaum suggests possibility of leukoplakia (because of white blebs) and importance in all macroglossias of serologic studies for syphilis.

188—Query: **Recurrent ulcers of the mouth.** J. A. M. A. **106**(22): 1936. May 30, 1936.

A woman twenty years old has had ulcers at intervals of two to three weeks for several years, requiring several days to develop and disappearing in four to five days. Of the multiple causes of ulcers of the oral mucosa the inquirer in this case has ruled out bad mouth hygiene, gastro-intestinal dysfunction, Vincent's infection, fungus infestation, pyorrhea, tonsils, reflex menstrual shock, and pernicious anemia. There remains the possibility of certain skin diseases, namely, herpes simplex, pemphigus, lichen planus, lupus erythematosus, and erythema multiforme, and the Query Editor mentions also the possibility of: (1) food sensitization; (2) laxative drugs containing phenol phthalein or other well-known chemicals to which many patients are sensitive; (3) possible electro-galvanism with deposition of ionized toxic metals into the oral mucosa (Lain). For food sensitization tests the elimination method is considered better than the skin scratch tests. Since this patient has dissimilar metal fillings, an electrical engineer with his delicate microammeter or galvanometer could determine the possibility of electro-galvanism as the cause of the stomatitis.

189—Rattner, Herbert, M.D.: **Stomatitis due to sensitization to dental plates,** in: J. A. M. A. **106**(26):2230. June 27, 1936.

Rattner calls attention to the fact that in susceptible individuals an irritant stomatitis may result from the wearing of dental plates made of some of the newer materials, synthetic substitutes for vulcanized rubber. The author recognizes the possibility of impaired circulation, mechanical injury from impingement, uncleanliness especially from poorly vulcanized porous dentures, and chemical irritants from pigments, etc., as causes of sore mouths. But he cites 3 interesting cases in which the stomatitis undoubtedly resulted from dental plates made of material to which the individual's mucous membranes were sensitive. The irritation commenced with wearing of the dentures, and symptoms increased until they were removed or replaced by vulcanite. In two of the cases (in both of which the plates were made of hecolite) a definite and positive skin reaction was obtained by strapping the plate to the inner surface of the arm. An illustration shows the positive skin patch to be of the horse-shoe shape of the denture which had rested upon it. No skin reaction followed the tests with vulcanite plates. These synthetic substances are made by processes which are trade secrets, but it is known that most of them are essentially cellulose. "Others consist of phenolresin compounds containing phenol-formaldehyde ammonia," etc., and it has been pointed out that "coloring with chrome, polishing with lime and the numerous liquids used to dissolve cellulose" can be ample sources of injury to mucous membranes. The cases reported confirm this.

190—Robbes, P.: **Vulcanite impression of mobile teeth,** in: Semaine Dentaire, **11**: 919. Jan., 1936.

The author describes a vulcanite apparatus made from an accurate impression of the incisal edges of the teeth concerned to be used in stabilizing mobile teeth during cleansing process. While the patient brushes the gingivae the vulcanite tray is bitten on thereby keeping the teeth in place. This helps establish occlusion and sets a position for the poorly adjusted position of the teeth.

191—Schonfeld, William, M.D.: **Phlegmon of the pterygo-palatine fossa following injury to hard palate with lollipop stick,** in: J. A. M. A. **108**(3): 194. Jan. 16, 1937.

Schonfeld warns that lollipops mounted on sharp sticks are as dangerous to young children as sharpened pencils. He describes at length the case of 22 months old child, in which an apparently insignificent injury to the hard palate near its junction with the soft palate terminated fatally. The infection spread via the pterygo-palatine canal to pterygo-palatine fossa with formation of a phlegmon and subsequently it spread to orbit, middle and cranial fossae. Orbital cellulitis, localized purulent meningitis, encephalitis and epidural abscess supervening; death followed 6 days later.

192—Vienna Dental Congress: (The Ninth International Dental Congress). Report of regular Vienna Correspondent, Aug. 14, 1936 in J. A. M. A. **107**(14): 1145-47. Oct. 3, 1936.

In the section on pyorrhea, Prof. Maurice Roy of Paris discussed the influence of hereditary defective arrangement on the

loosening of teeth. Prof. Entin of Leningrad said the formation of pus pockets is not well understood, but disturbances of metabolism and of glands of internal secretion and the condition of the gastro-intestinal tract as well as disturbances of the nervous system figure in its etiology: disorders of kidneys and liver are frequently coincident with dental disturbances. Haüpel of Prague believes that any general illness influenced by cellular metabolism is capable of producing pyorrhea and that it may be a sequence to infectious disease, notably influenza; he finds it more frequent in slender than in stout individuals; calculus and other local factors are to be considered. Haüpel recommends the application of oxygen and escharotics, and eradication of pus pockets, but to restore gums to health adequate hygiene is also necessary. Weinmann of Vienna has noticed hypofunction of the anterior lobe of the hypophysis in pyorrheal patients; Haüpel has often observed that Doppler's operation (on the sympathetic nerves in the region of the gonads) leads with experimental certainty to amelioration of the purulent and loosening process. Schour of Chicago also has found this to be true. Orban of Vienna reiterated the belief of that school that there are two forms of pyorrhea alveolaris, one due solely to poor hygiene, the other to disturbances of general metabolism. Any course of treatment must be accompanied by removal of tartar and prompt correction of dental defects, and by careful instructions in toothbrushing. At best, said Dr. Orban "the treatment of pyorrhea will require an unusual amount of time and patience."

193—Williams, Herbert Ely, D.D.S.: **"Standing vs. sitting,"** in: J. A. D. A. **23**: 627-628. April, 1936.

The author criticizes the habit of some dentists of standing while operating and claims that they burn more energy, that their bodies are not balanced, and that they are consequently more fatigued at the end of the day than those dentists who have remained seated while operating. It is not according to Nature to remain standing for such long intervals of time and it detracts from the steadiness and accuracy which are essential to good work.

—*G. R. S.*

Twenty-Fifth Annual Meeting

AMERICAN ACADEMY OF PERIODONTOLOGY

SAINT LOUIS, MISSOURI

OCTOBER 20, 21, 22, 1938

THURSDAY

Morning

REPORTS OF OFFICERS AND COMMITTEES:

Secretary-Treasurer. Raymond E. Johnson.

Committee on program. Grace Rogers Spalding.

Committee on clinics. Joseph R. Walsh.

Committee on general arrangements. J. A. Jacobsmeyer.

Committee on nomenclature. Arthur H. Merritt.

Committee on publication. Harold J. Leonard.

Committee on scientific investigation. (Report Friday Morning.)

Special committee reports. Harry J. Lyons, Justin D. Towner, Harold J. Leonard, Lynn Fonner, Wilia Yeretsky, Carlos H. Schott, C. J. Caraballo.

Presidential address. Clayton H. Gracey.

Afternoon

THE HISTORICAL BACKGROUND OF PERIODONTIA:

The history of oral prophylaxis. Willa Yeretsky, Grand Rapids, Michigan.

The influence of type and design of instruments on the development of interest in periodontal diseases. John Oppie McCall, New York City.

Progress in the recognition of etiologic factors of periodontal diseases. A. W. Bryan, Iowa City, Iowa.

Diagnosis and treatment of periodontal diseases. Harold J. Leonard, New York City.

History of Vincent's infection. Isador Hirschfeld, New York City.

Bibliography. Samuel R. Parks, Dallas, Texas.

Evening

BIOGRAPHICAL SKETCHES

Arthur H. Merritt, New York City.

Formal Reception

FRIDAY

Morning

Past-presidents' breakfast.

Report of past-presidents' meeting. Carlos H. Schott, Chairman.

The past and future of periodontia. Paul R. Stillman, Longwood, Florida.

Report of committee on scientific investigation. Edgar D. Coolidge, Chairman.

What happens to the supporting structures in traumatic occlusion. Balint Orban, Chicago, Illinois.

Presentation of a plan for clinical research by Academy members. Edgar D. Coolidge.

DISCUSSION LUNCHEON

Leaders: Rudolf Kronfeld and Lynn Fonner

1. What important advancements in diagnosis and treatment of periodontal diseases have been made since the founding of the Academy? Edward B. Spalding.
2. Is there any record of well-observed, well-controlled cases of periodontal diseases having been benefitted from the administration of calcium, phosphorus, or vitamin preparations? Carlos H. Schott.
3. Of what value is a mouth smear in Vincent's infection or other oral lesions. Harold J. Leonard.
4. How should executives of dental colleges be approached to secure more instruction in periodontia? Samuel R. Parks.
5. Is it possible to standardize the treatment of Vincent's infection? If so, state recommendations. J. A. Jacobsmeyer.
6. Electro-coagulation versus surgery in the treatment of pyorrhea pockets, from patient's and the operator's point of view. Olin Kirkland.
7. If a bridge is indicated in a mouth with advanced, yet treated, periodontoclasia, which type is preferable, fixed or removable? M. Harry Garvin.
8. Of what value is a complete physical examination to periodontal treatment? Detail. Dickson G. Bell.
9. The periodontist as a husband. Mrs. Clyde C. Sherwood.

Afternoon

TABLE CLINICS AND EXHIBITS

Clinics and Historical Exhibits

Joseph R. Walsh, Chairman

1. Austin F. James, Chicago, Illinois. Study models of cases where all natural teeth are present showing no caries, but the development of traumatic occlusion and early alveolar bone resorption. Models of same cases shown after relief of occlusion.
2. Isador Hirschfeld and Frank E. Beube, New York City. The development of the tooth brush and oral hygiene. (Historical exhibit and table clinic)
3. Carlos H. Schott, Cincinnati, Ohio. Splints for loose teeth (Historical).
4. C. W. Hoffer, Nashville, Tenn., Bridge abutments and the positioning of their gingival margins for the prevention of periodontal lesions and the maintenance of healthy gingivae around these abutment teeth.
5. T. E. Braly, Chattanooga, Tenn., Models and x-rays of mouth during and after reconstruction.
6. Bernard D. Friedman, Chicago, Illinois. The practical treatment of periodontoclasia.
7. Hunter S. Allen, Birmingham, Ala. "Let's moor closer to earth."
8. Arvah S. Hopkins, Gulfport, Miss., Results from the conservative treatment of pyorrhea.
9. Lillian Barkann, New York City. A conservative surgical technique for the eradication of a pyorrhea pocket under topical anaesthesia.
10. J. A. Jacobsmeyer, St. Louis, Mo. The importance and methods of altering stress on occlusal surfaces.

Evening

ANNUAL BANQUET

SATURDAY

Morning

Radiodontic services in periodontal practice. Clarence O. Simpson, St. Louis.

A new treatment for the interproximal space and septal tissue. James M. Prime, Omaha, Nebraska.

Election of officers.

Luncheon meeting of new council.

Afternoon

Informal tea for members and guests.

INDEX OF ABSTRACTS

Bold faced figures refer to abstracts, light face figures to pages.

Alkaline reserve in diagnosis of pyorrhea, 279-102.
Allergic manifestations in the oral mucosa, 289-105.
Alveolar absorption of syphilitic origin, 364-95.
Alveolar atrophy in anemic dogs, 187-88.
Alveolar bone atrophy, 369-97.
Alveolodental ligaments, 370-97.
Alveolar disease with deep pockets, 287-105.
Antiseptics in aqueous solution, *in vitro* activity of, 466-108.

Bacteria, effect of oral mucosa on, 75-90.
Bacteria, effect of saliva on, 74-90; 77-90.
Bacteriology of periodontoclasia, 78-90.
Betel chewing in relation to oral sepsis and carcinoma, 168-113.
Biologic equilibrium in a dentition where some teeth are already extracted, 167-113.
Blood dyscrasias, differential oral diagnosis in, 290-105.
Bone changes following conservative periodontal treatment, 468-108.
Buccodental manifestations of infantile acrodynia, 285-104.

Conjunctivitis due to fusospirochetal infection, 173-115.

Dental caries and paradentosis in Brazil, 177-116.
Dental caries and periodontal disease, 375-99.
Dental examination of inhabitants of Tristan Da Cunha, 367-96.
Diabetes and paradentoses, 288-105.
Dryness of mouth, 181-116.

Epithelial attachment, biological and mechanical significance of, 75-93.
Equilibrium of the articulation, 383-102.

Fixation of the teeth in treatment of paradentosis, 464-107.

Garlic breath odor, 169-114; 175-115.
Gingival inflammations, micrology of, 76-90.
Gingival massage, 64-110.
Gingival tuberculosis, 280-103.
Gingivectomy, 471-109.
Gingivitides and oral spirochetoses, 66-111.
Gingivo-dental affections in pathology of the skin, 281-103.

Histologic analysis of child's jaws in malocclusion, 77-93.

Injuries to the teeth and supporting structures, 382-101.
Injury to hard palate with lollipop stick, 191-118.

Knorpel in periodontium, 378-100.

Leukoplakia, etiology, pathology and treatment of, 178-116.

Mouth brushing, importance of, 65-110.

Oral and gastric flora, 79-90.
Oral and systemic disorders, 282-103.
Oral flora, effects of medicaments on, 80-91.
Operative dentistry, failing? 180-116.
Osteofibrosis, treatment of, 469-108.

Paradental disease with splenomegaly, 171-114.
Paradentosis, 81-91; 286-104; 365-95; 368-96; 377-100.
Paradentosis and gingivitis, health reactions to treatment of, 460-106.
Paradentosis, application of law of Koelliker to, 380-101.
Paradentosis, contribution to the therapy of, 472-109.
Paradentosis, etiologic stimuli, 371-98; 376-99.
Paradentosis from the standpoint of general medicine, 470-109.
Paradentosis, scientific research on, 80-94.
Paradentosis, surgery of, 462-107.
Paradentosis, treatment by intravenous injections of flouride of calcium in a suspension of magnesium, 467-108.
Parodontal disease, classification and histopathology of, 188-88.
Pathologic tissue changes in pulp and paradentium, 186-88.
Periodontoclasia, 373-98; 374-98.
Periodontoclasia and allergic oral derangements, 384-102.
Periodontoclasia and gingivitis, medical aspects of, 176-115.
Periodontoclasia and the glands of internal secretion, 283-103.
Periodontoclasia, etiology of, 381-101.
Periodontoclasia, metabolism in, 71-92.
Periodontoclasia, relation between general disease and, 372-98.
Phosphatase, study of, 78-93.
Podophyllum, sensitivity to, 183-117.
Potassium chlorate in dentifrices, 174-115.
Pseudocolloid of the lips, 185-117.
Pyorrhea alveolaris, 284-104; 379-100.

Report at National Dental Congress, 291-106.
Resting dentition outside of meals, 463-107.

Saliva of Kuskokwin Eskimos, 76-93.
Saliva, retention of reducing sugars in, 82-95.
Salivary antiseptic, 72-92.
Salivary calculus, 81-95.
Salivation, 182-117.
Scurvy, experimental, 70-91.
Sodium metaphosphate, 74-92.
Sore mouth in nephritis, causes of, 170-114.
Stabilization of loose teeth, 461-107.
Standing vs. sitting, 193-119.
Stomatitis due to sensitization to dental plates, 189-118.
Sugar and cereals, comparison of crude and refined, 79-94.
Systemic and blood picture findings in periclasia-free and periclasia-involved individuals, 73-92.

Tartar accretion, 366-96.
Tongue burning, 186-117; 187-117.
Tongue, painful, 184-117.
Toothbrush, advancement of, 63-109.
Treatment of inflammatory conditions with Selenium colloidale, 465-107.
Tuberculous ulceration of mouth and pharynx, 179-116.

Ulcers of the mouth, recurrent, 188-118.

Vienna Dental Congress, 192-118.
Vincent's infection, 221-111; 222-111.
Vincent's infection, diagnosis, treatment, and prevention, 225-112; 226-113.
Vincent's infection, misconception of, 227-113.
Vincent's infection, public health significance of, 223-112.
Vincent's infection, treatment with acetarsone, 224-112.
Vincent's stomatitis and granulocytopenia, 220-111.
Vitamin D, new forms and sources of, 189-89.
Vitamin deficiency, 189-89.
Vitamin therapy, 172-114.
Vulcanite impression of mobile teeth, 190-118.

Index of Authors

Abdala, Dr. Salvador ---- 95
Adloff, P. ---- 113
Aksoy, Z. C. B. ---- 102
Albert, Leo ---- 109
Appleton, J. L. T. ---- 90
Ayllom, Pedro ---- 95
Badanes, Bernard B. ---- 96, 110
Badeff, T. ---- 92
Balendra, W. ---- 113
Ball, Rita P. ---- 90
Barnes, H. N. V. ---- 96
Bert, René ---- 108
Bessey, O. A. ---- 97
Beust, Theo. B. ---- 90
Bibby, Basil G. ---- 90
Blankenhorn, M. A. ---- 114
Blessing, Georg ---- 96
Bliss, S. ---- 114
Boyens, Paul J. ---- 106
Boyle, Paul E. ---- 91, 97
Breuer, Karl ---- 114
Bricker, Frederick A. ---- 110
Brinch, Ove ---- 88
Chatellier, Jean ---- 97
Christie, David ---- 111
Clark, D. R. ---- 111
Dailey, F. H. ---- 115
Davidoff ---- 98
De Libouton ---- 107
Deliberos, J. ---- 103
Dietz, Anne K. ---- 90
Dillon, Juan ---- 107
Dunnington, J. H. ---- 115
Farrell, G. W. ---- 111
Fosdick, Leonard S. ---- 95
Goldman, Henry M. ---- 88
Goldstein, Harold ---- 92
Gordon, Samuel M. ---- 92
Gottlieb, Bernhard ---- 107
Greenbaum, Sigmund ---- 117
Grove, O. H. ---- 111
Grythe, Olaf ---- 90
Haggard, Howard W. ---- 115
Hall, J. Frank ---- 88
Hatton, Edward B. ---- 93
Healy, J. C. ---- 115
Holst ---- 98
Hood, M. ---- 90
Iskraut, H. ---- 98
Jarlow ---- 98
Karshan, Maxwell ---- 93, 95
Khorazo, D. ---- 115
Koch, F. ---- 116
Kohler, G. ---- 107
Kronfeld, R. ---- 93
Labbé, Gonzalés ---- 93
Le Norcy ---- 103
Linka ---- 98
Lyons, D. C. ---- 112
Mahé, Gustav ---- 99
Marichal, B. ---- 98
Martin, David J. ---- 104
Maxwell, C. H. ---- 112
McCarthy, F. P. ---- 116
McNichols, W. A. ---- 111
Nesson, J. H. ---- 112
Nielsen ---- 100
Nivard ---- 107
Noriskin, J. N. ---- 94
Nye, R. N. ---- 108
Orban, B. ---- 100
Ormerod, F. C. ---- 116
Osborn, T. W. B. ---- 94
Parodneck, Clara B. ---- 96
Pierron ---- 108
Prime, James Mark ---- 116
Rachet, Marcel ---- 104
Rattner, Herbert ---- 118
Richards, C. E. ---- 114
Robbes, P. ---- 118
Robinson, Hamilton B. G. ---- 38
Rose, E. D. ---- 113
Rosebury, Theodor ---- 98
Rosenthal, S. Leonard ---- 91
Rosenthal ---- 108
Roy, M. ---- 100
Rustishauser, E. ---- 101
Schilling, Viktor ---- 104
Schonfeld, William ---- 118
Siegmund, Herbert ---- 94, 101
Sorrin, Sidney ---- 108
Stas, J. ---- 94
Stuteville, O. H. ---- 101
Sweet, Marion H. ---- 115
Szabo, J. ---- 109
Tenenbaum, Benjamin ---- 95
Teuscher, George W. ---- 95
Thibault, R. ---- 109
Thielemann, K. ---- 102
Thoma, Kurt H. ---- 88
Tishler, B. ---- 113
Trauner, Richard ---- 105
Vantier, U. ---- 109
Van Vouno, N. C. ---- 102
Weichert, Johanna ---- 105
Weiss, M. ---- 91
White, Cleveland J. ---- 105
Williams, Herbert Ely ---- 119
Winter, Leo ---- 105
Wissotsky ---- 106
Wolbach, S. B. ---- 89, 97

INDEX OF JOURNAL OF PERIODONTOLOGY, VOL. 9, 1938

January

		Page
Gingival and Periodontal Disease in Children	JOHN OPPIE MCCALL	7
Infectious Mononucleosis	JAMES E. COTTRELL	15
Report of a Study on the Etiology of Bleeding Gums	RUDOLPH H. PELZER	25
Salivary Calculus	THEODOR ROSEBURY	40
Salivary Analysis of Patients Subject to Wastings: Abrasions and Erosions—Frances Krasnow, Edith B. Oblatt, Guggenheim Dental Clinic; and Isador Hirschfeld		42
Factors Influencing the Kinds and Numbers of Mouth Bacteria	J. L. T. APPLETON	46
Coöperation of the General Practitioner, Periodontist and Patient	JUSTIN D. TOWNER	48
A Board of Periodontology	H. J. L.	49
Certification of Periodontists	EDWARD B. SPALDING	51
Resumé of Report of Pathologic Conditions Associated with the Use of Sodium Perborate, Observed by Members of the American Academy of Periodontology	ISADOR HIRSCHFELD	54
The Quarter Century Mark	CLAYTON H. GRACEY	54
Case Reports		56
Apparent Selection of Similar Areas Other Than the Gingival Crevice by Spirochetes and Fusiform Bacilli in Five Related Cases of Infection	BENJAMIN TISHLER	56
Stomatitis—Caused by Chemical Burns Due to the Use of Pure Sodium Perborate	MARTHA E. JONES	57
Endocrine Therapy in Atrophic Periodontoclasia	WILLA YERETSKY	59
Localized Hypertrophic Gingivitis Due to Tongue Habit	JAMES E. AIGUIER	59
"Doctor, What Dentifrice Do You Recommend?"		60
Book Reviews		61
Program—Twenty-fifth Annual Meeting A. A. P.		63

July

Advantages and Disadvantages of Surgical Periodontia	EDWARD L. BALL	71
Subgingival Curettage	R. W. RULE	77
Evaluation of the Oral Smear in Vincent's Infection	JEROME S. GROSBY	85
The Other Side of the Story		87
Abstracts and Extracts of Current Dental Literature for 1937		
(a) Periodontal Histo-pathology		88
(b) Periodontal Bacteriology		90
(c) Other Laboratory Researches		91
(d) Clinical Etiology		95
(e) Symptomatology and Diagnosis		102
(f) Treatment		106
(g) Home Care and Prophylaxis		109
(h) Vincent's Infection		111
(i) Miscellaneous		113
Twenty-Fifth Annual Meeting		119
Index of Abstracts		121
Index of Authors		122